list

ATTACHMENT H

Evaluation of EPA's Monte Carlo and Microexposure Event Analyses of the Fish and Waterfowl Consumption Scenarios

1.0 Introduction

The Human Health Risk Assessment (HHRA) for the Housatonic River includes both a simple Monte Carlo Analysis (MCA) and a Microexposure Event (MEE) analysis for each of the fish and waterfowl consumption risk assessments. While EPA has undertaken these analyses in an effort to provide risk managers with a more complete evaluation of potential risks due to these exposure pathways, there are a number of problems with the approach that has been used. The net result is that the analyses presented by EPA do not accurately reflect the data upon which they are based or the relationships among the variables. As a result, the outputs from these analyses are essentially the same as the results of the point estimate calculations and do not constitute an improvement over point estimates for characterizing potential risks to consumers of fish or waterfowl in the HHRA.

This Attachment provides comments on the MCA and MEE analyses that have been conducted as part of the HHRA and suggests improvements that can be made to further refine the model structure, its input parameters, and the model outputs. In addition, based on these suggested changes, GE provides an alternative MEE analysis for fish consumption, which incorporates the recommended changes and demonstrates the difference in output from the model when the recommended modifications are made.

2.0 Evaluation of EPA's Probabilistic Analyses

Probabilistic models like MCA and MEE have the capability of incorporating the full range of data available for each exposure parameter, thereby avoiding the need to summarize those distributions and select a single, questionably representative, summary statistic, as is done in the point estimate approach. These models can also easily incorporate relationships among the input variables and their strength depends on the recognition and inclusion of these important relationships. Robust and critical evaluations, performed using either Monte Carlo or MEE modeling, require the use of data, distributions, and paradigms that are representative of the exposure scenario under evaluation. Reliable data exist to provide discrete distributions for

most of the variables in the risk equations for fish and waterfowl consumption, and these data should be directly used. Consideration of data should be sufficient to allow the retention of discrete data as probability mass functions when appropriate. In addition, the selection of distribution types and defining values should not be based on summary statistics, unless those are the only values available, but should instead include the full range of the data, without assumptions about the shapes of their underlying distributions, thereby refining risk results instead of simply duplicating point estimates (see EPA, 2001, Appendix B).

MEE analysis is expected to produce different results from both a point estimate analysis and a simple MCA (Price et al., 1996; Simon, 1999; EPA, 2001) because exposures are modeled as a series of separate exposure events. It allows variations in each individual's behavior to be modeled, considering both differences that occur from event to event, as well as differences that occur over time. Instead of focusing on the exposure of an entire population, as is done in a deterministic approach, MEE permits the estimation of the variation of exposures for individuals within the exposed population. The more refined and representative the model, the better the estimation of risks. While the calculated values from the less refined models, like the point estimate and MCA, will be found within the output distributions from the MEE, the model will provide a more robust representation of the wide range of potential exposures and risks that will occur because it will allow nearly limitless combinations of input parameters to be evaluated and quantified.

The following sections summarize GE's principal concerns with the MCA and MEE analyses used in the HHRA and recommend ways to improve those analyses so that the results better reflect actual exposures via fish and waterfowl consumption.

2.1 Failure To Use Distributions for Exposure Point Concentrations

While a substantial set of discrete data is readily available for fish tissue concentrations, EPA has chosen instead to derive an upper bound PCB concentration – i.e. the 95% upper confidence limit (95% UCL) on the mean -- for each river reach, based on those data, and use it as a single input to both the MCA and MEE models (HHRA, Vol. IV, Tables 4-5, 4-6, 4-7 and 6-

2).[1] For the reaches where both the bass/bullhead and sunfish/perch species groupings were combined for evaluation, EPA derived a 95% UCL for the bass/bullhead group, a separate 95% UCL for the sunfish/perch group, and then averaged the two 95% UCLs to get the exposure point concentration (EPC) that was actually used in the exposure and risk estimates. This provided a single estimate of PCB concentrations that is the same as, and therefore no more refined than, the EPC used in the point estimate.

Anglers will not always catch and eat fish with a 95% UCL concentration but will, in fact, eat fish of varying concentrations over time. To avoid the kind of bias that is introduced by reducing a large body of data down to a single, and likely biased, point estimate, EPA can and should use all the fish tissue data in the input distribution. This would require no manipulation of the data and would allow the EPC for each fish meal eaten to be selected, at random, from the existing distribution of fish tissue data. This most closely approximates potential exposure to an individual over time because the actual fish tissue concentration in each fish harvested from the Housatonic River would have an equal likelihood of occurrence. Use of the full distribution of data would thus greatly improve the model's ability to approximate actual exposures.

EPA has used a similar approach for the waterfowl consumption analysis. Instead of incorporating all of the available duck tissue data into the model, it has reduced the available sampling data to a single statistical point (the 95% UCL) and used it as the EPC (Vol. IV, p. 6-49). This value, which is the same as the EPC used for the point estimate analysis, obscures the variability within the sampling data and does not reflect the variability in concentrations to which waterfowl consumers will be exposed during individual meals. Like fish consumers, the waterfowl consumers will consume birds that have variable concentrations over time. Thus, it would be far better to retain the natural variation within the data for the MEE analysis rather than use a summary statistic as a point estimate.

2.2 Selection of Fish Consumption Rates

EPA has converted the raw fish consumption data from the Ebert et al. (1993) study in Maine into a frequency of meals per year using an estimated meal size, and then used this range of

[1] It should be noted that while Table 6-2 indicates that the arithmetic mean was used as the EPC for the MCA and MEE analyses, this statement is in error. The EPCs used were identical to the combined 95% UCL EPCs used in the point estimate analysis, as shown in Tables 4-5, 4-6, and 4-7.

meal frequencies as an input distribution for the probabilistic analyses (HHRA, Vol. IV, p. 6-20). In doing so, EPA has used the consumption rates calculated by Ebert et al. (1993) for "all waters" assuming "no sharing." As shown in Section 4.2.1 and Attachment G of these comments, it is not appropriate to use either the "all waters" or "no sharing" consumption data in the HHRA as these are not reflective of actual conditions. The same applies to the probabilistic analyses.

In addition, EPA has expanded the derived frequency ranges to include upper probability bounds, well above the actual reported ranges. As discussed in the next section of this Attachment, this expansion of the distribution to include conjectural upper bounds is both unnecessary and inappropriate.

2.3 Expansion of Distributions To Include Hypothetical Upper Bounds

In an effort to ensure that input parameter distributions do not exclude any possible values, EPA has intentionally incorporated additional levels of conservatism into the input distributions by expanding the actual distributions to include calculated upper bound estimates. This has been done both for fish consumption rates and for waterfowl meal size (HHRA, Vol. IV, pp. 6-19 to 6-22). These added levels of conservatism are not based on empirical data but are instead statistical conjectures about possible upper bounds for the input parameters. The end result consists of highly conservative estimates of risks and hazards that are based, in part, on conjectural values and are no more refined or accurate than the point estimates with which the Agency began.

It is not appropriate to intentionally introduce additional layers of conservatism into the model by "expanding" the distributions when there are already solid input distributions available for the parameters in question. To do so results in implausible combinations of parameters that are no longer based on the data. For example, for the fish consumption model, EPA has established a hypothetical maximum fish meal frequency of 1,042 half-pound fish meals per year (equivalent to 2.9 half-pound meals per day every day) from a single reach of the river every year for 70 years (Vol. IV, p. 6-13). EPA has then further "fattened" that new distribution by an artificial uncertainty bound of 10 percent (Vol. IV, p. 6-14), thereby raising the hypothetical maximum by an additional 10 percent – to 1,146 fish meals per year, which equates to more than three half-pound fish meals per day from a single river reach every day for 70 years. The level of fishing

effort necessary to provide three half-pound meals daily throughout the year makes this estimate wholly implausible.[2]

In addition, this bounding estimate largely ignores the data upon which the input distribution is based. In the Ebert et al. (1993) survey, which is used as the basis for the fish consumption rate input distributions, consumption rates were reported for 1,007 survey respondents who consumed fish. The maximum fish consumption rate reported (for all waterbody types combined) was 182 g/day (approximately 6.4 ounces/day). This equates to roughly one fish meal daily throughout the year and represents a plausible but conservative upper bound, particularly for a single river reach. It is consistent with both the maximum values reported in other surveys of sport-caught freshwater fish consumption (Pao et al., 1982; West et al.; 1989; Connelly et al., 1992, 1996; Ebert et al., 2002), discussed in EPA's *Exposure Factors Handbook,* and with EPA's recommendation for evaluating upper percentile fish ingestion by subsistence populations (EPA, 1997a). Thus, there is no reason to assume that the Maine angler survey data are not representative and to include other conjectural values into the input distribution.

Similarly, EPA has expanded the meal size distribution for the waterfowl consumption scenario, apparently based on its own extrapolation of the data. In doing so, EPA has used a hypothetical upper bound estimate of 675 g/meal (1.5 pounds of duck per meal), along with a central estimate of 188 g/meal and a lower bound estimate of 1 g/meal – all purportedly based on poultry meal sizes reported by Pao et al. (1982), as cited in EPA's (1997a) *Exposure Factors Handbook* (see HHRA, Vol. IV., p. 6-50). However, Table 11-23 of the latter document, which reports the findings of Pao et al. (1982), provides no absolute maximum meal size. It lists the 99th percentile meal size as 388 g, the average and median meal sizes as 128 g and 112 g, respectively, and the 5th percentile meal size as 42 g. Thus, EPA's hypothetical meal size distribution no longer resembles the empirical distribution reported by Pao et al. EPA's maximum meal size of 675 g and its central estimate of 188 g both substantially overstate the empirically based values and thus introduce unnecessary bias into the exposure estimates.

[2] To obtain 3 half-pound meals of edible fish daily, one would have to harvest 4.7 pounds of whole fish from the river daily, every day of the year. According to the Housatonic River creel survey conducted by CTDEP (1986), for example, the average catch rate in Lake Lillinonah was 2.28 fish/hour. If fish caught average ½ pound in size, this catch rate indicates that an angler would have to fish approximately 4 hours per day every day throughout the year in order to catch sufficient fish to support this ingestion rate. In other reaches of the river in Connecticut, where catch rates ranged from 0.97 to 1.6 fish/hour, effort would need to be as high as 10 hours per day, every day of the year. This level of effort is not plausible.

EPA's addition of non-empirical, conservative values to the input distributions is also not consistent with Agency recommendations. An EPA (1999a) report on selecting input distributions for probabilistic assessment states: "After some additional discussion, it appears that the experts were in agreement that one should strive primarily for accuracy and that ideally any adjustments that introduce 'conservatism' should be left to decision makers." Therefore, the introduction of extrapolations of extreme values in a distribution, as have been incorporated in the HHRA's probabilistic models, should be avoided. Use of the full range of the input distributions will allow combinations of upper bound parameters to occur so that the full range of possible risks are included in the output. Ultimately, the decision of which percentile in the final risk distribution is sufficiently protective is a risk management decision, but the point of departure is generally the 95th percentile (EPA, 2001).

Finally, it should be noted that EPA included these upper bound estimates without making any adjustment of the extreme value frequencies. For example, the Ebert et al. (1993) data for all waters combined included approximately 1,000 individuals. EPA has added an upper bound value of 1,146 meals, artificially indicating that the probability that this meal frequency will occur is roughly 1 in 1,000. Assuming that it is even plausible that someone would eat this many fish meals annually, it might require a survey sample on the order of 50,000 anglers in order to find that individual. If this were the case, the probability that this value would occur would be on the order of 1 in 50,000 rather than 1 in 1,000 because roughly 49,000 additional data points would be added to the distribution. Thus, EPA has contributed additional upper bound values without correcting for the probability that they will occur. If one changes the number of elements in a distribution, without altering its shape, the results will be inappropriately skewed and will no longer be representative. To ignore the relative frequency of the maximum input inappropriately increases the impact of the extreme value, which is no more likely to occur regardless of the number of elements in the distribution. In fact, it is likely that an exponential decline of probabilities will occur as the upper boundary is approached.

2.4 Lack of Adequate Correlation Among Model Inputs

EPA has also assumed that all exposure events are independent of each other. However, there are several factors that are not independent from event to event. The relationships among

these individual events impact the final risk distributions and should be considered. These include, but are not limited to, the following.

- Consumption rates -- Consumption rates may fluctuate from year to year but are likely to remain similar over time. For example, an individual who consumes fish frequently in one year is likely to consume fish frequently in other years. While the actual rate of fish consumption by an individual will vary somewhat from year to year due to differences in effort and success over time, it is likely that consumption rates will remain similar over time. It is not likely that someone who is an infrequent consumer of sport-caught fish one year will be a high consumer the next (EPA, 2001). EPA's analysis, however, makes it equally likely that the person who consumes the minimum amount of fish one year will consume the maximum amount the following year. As a result of adopting EPA's assumptions, over time, each individual's consumption rate approximates the average value and the consumption rates selected for individual anglers over time are very similar to the point estimate value used in the deterministic analysis. Hence, exposure and risk estimates are similar between the two analyses. Through the use of this model, not only are risks overestimated for a large segment of the angling population, but the true shape of the risk distribution is lost as well. This results in a flattening of the risk distribution such that potential risks to high-end fish consumers would be underestimated, were it not for the artificial introduction of overcompensating factors elsewhere in the analysis.

- Body weights -- EPA's MCA and MEE analyses select a single body weight to evaluate exposure over the entire exposure duration (HHRA, Vol. IV. pp. 6-24 to 6-26). This is not reflective of actual conditions. The change in body weight that occurs as individuals age is a well-documented phenomenon (EPA, 1997a). In addition, body weight is affected by the gender of the exposed individual and should be considered in the selection of input values.

- Cooking losses – In EPA's MCA and MEE models, cooking loss values were estimated and weighted by cooking preference, and the calculated weighted averages were then used as inputs to the models (HHRA, Vol. IV. pp. 6-15 to 6-18). This approach does not differentiate between the cooking methods used to prepare individual species. Cooking loss is a function of the cooking method used, and the cooking method used is largely a

function of the species and size of fish that are consumed. Hence, it is important to tie cooking loss to the fish species.

There are substantial data available on fish cooking methods used by recreational anglers as well as species-specific cooking method data (e.g., Connelly et al., 1996; Ebert et al., 1993 unpublished data provided to EPA; Ebert et al., 2002). These data can be used to develop probabilities that certain cooking methods will be used, depending upon the species being consumed at each meal. In the MEE model, the species of each fish meal consumed can be identified. Once the species has been identified, a cooking method can be selected from the probabilities of each cooking method for that species; then the appropriate cooking loss factor can be applied for that cooking method.

In addition, the HHRA considers only three possible cooking methods (broiling, boiling, and frying) (HHRA, Vol. IV. pp. 6-15, 6-17) and uses data from limited number of studies to derive its cooking loss values. There are, however, additional peer-reviewed data available on cooking losses after frying low lipid level fish (Skea et al, 1981; Puffer and Gossett, 1983) as well as data on cooking losses that result from baking fish (Smith, 1972; Smith et al., 1973; Skea et al., 1981). All of these studies should be considered in deriving representative cooking loss factors.

2.5 Development of an Exposure Duration Estimate Based on MADPH Survey Data

EPA's current MCA and MEE models use distributions of exposure duration based on MADPH survey data on the amount of time respondents reported eating freshwater fish from any source (HHRA, Vol. IV, pp. 6-22, 4-56). However, since these responses related to the consumption of freshwater fish from *any source* (including store-bought fish and sport fish from other waterbodies), they do not provide information on the length of time that individuals have consumed sport-caught fish, let alone game, from the Housatonic River. It is possible that some information bearing on the length of time that people have eaten fish or game from the Housatonic may be obtained from fishing or hunting license information or other state recreational sources. In the absence of such information, it would seem more relevant, but still very conservative, to assume that individuals who catch or shoot and consume sport-caught fish or game from the Housatonic River may do so during each year that they live near the river. Exposure durations could thus be estimated using census data for the appropriate counties, taking into consideration population mobility and mortality rates.

2.6 Unfounded Concern About Underestimation of Exposures in MEE

EPA has indicated its concern about underestimating risks by using the MEE approach. The HHRA states: "The MEE modeling removes the possibility that some individual will be simulated who eats the maximum amount of the most contaminated fish and waterfowl at every meal for an entire lifetime. . . . This raises the possibility that some individuals who eat larger-than-average meals of more-contaminated-than-average fish and waterfowl more often than would be expected purely by chance are not represented in the model results." (HHRA, Vol. IV, p. 6-8.)

While EPA's concern may be true of the model that has been developed for this HHRA, a properly designed and executed MEE analysis will not be subject to this concern. By including a full distribution of the data in a properly designed MEE model, the so-called maximum consumer will be represented proportionally in the model output. That hypothetical individual would be present but at a frequency that is determined by the data sets. If the probabilities are vanishingly small for the occurrence of such a consumer, then the number of iterations that will be required for inclusion of that individual may be extremely large, but will occur if enough model iterations are completed. In this case, however, the 95th percentile value will not change significantly (the data will produce a representative output at that percentile from many fewer iterations) as the number of iterations skyrockets to fill in the upper boundary. The concern about excluding some individuals who eat larger-than-average meals of more-contaminated-than-average fish and waterfowl is also unfounded. An MEE model can allow an individual to maintain a high level of consumption from year to year, thereby ensuring a continuation of consumption patterns.

2.7 Lack of Consideration of Uncertainties in Dose Response

The above sections have focused on the limitations of EPA's probabilistic analyses insofar as they affect the exposure estimates. However, there is an additional, and potentially greater, source of uncertainty in the risk assessment that can be characterized in a probabilistic analysis – namely, the uncertainties associated with the dose-response values used to estimate risks. EPA's probabilistic analyses do not attempt to take account of these uncertainties.

Toxicity values based on human epidemiological studies are not available for PCBs. Thus, data from studies of laboratory animals provide the basis for these values. The practice of extrapolating effects observed in experimental animals to predict human toxic response to chemicals is a major source of uncertainty in risk estimates (EPA, 1989) because of the multiple uncertainty factors that are used in the extrapolation. The magnitude of the combined uncertainty around the toxicity values can be characterized in a probabilistic analysis. Several authors have provided information on the quantitative characterization of the uncertainties in toxicity dose-response values (Evans et al., 1994a,b; Baird et al., 1996; Slob and Pieters, 1997; Swartout et al., 1998; Crouch et al., 2001). For example, Swartout et al. (1998) describe an approach in which, to demonstrate the uncertainty associated with the non-cancer Reference Dose (RfD), the equation for setting the RfD can be used, replacing the point estimate uncertainty factors in the RfD with distributions. A probabilistic technique can then be used to determine the uncertainty in the estimated RfD. Such quantitative consideration of uncertainty in dose response is consistent with recommendations of EPA's Science Advisory Panel (SAP) under FIFRA. In February 1999, the SAP reviewed EPA's proposed approach for assessing non-carcinogenic risks from aggregate exposure to pesticides (EPA, 1999b) and called for the use of quantitative techniques for the evaluation of uncertainty in non-carcinogenic and carcinogenic risks as a means of improving EPA decision-making, stating that "it would like to see the whole NOAEL/uncertainty factor framework replaced by a more quantitative risk assessment approach in which all of the safety factors are replaced by distributions based on the best available data from well studied cases." (EPA, 1999b, p. 37)

Although GE recognizes that EPA's current guidance on probabilistic risk assessments (EPA, 2001) does not provide for the use of distributions for the toxicity values, GE believes that the probabilistic analyses conducted in the HHRA would be greatly improved if they included, at least as a sensitivity analysis, a quantitative evaluation of the uncertainties associated with the selected dose-response values. As an example, GE has included such an analysis in Exhibit H.1, with further details provided in Exhibit H.2, as discussed further below.

2.8 Lack of Transparency in the Analyses

The HHRA lacks sufficient transparency to comport with EPA's probabilistic risk assessment guidance (EPA, 2001) and its information quality guidelines (EPA, 2002). The discussion of the MCA and MEE is highly technical. It is difficult to understand the degree to which the "fattened"

data (i.e., the expanded distributions to include hypothetical upper bounds) impact the analysis or to determine how the models actually work. Analyses and related graphics should differentiate, illustrate, and compare the results that EPA derived using the empirical data with the data sets that have been augmented. Comparing the "fattened" data set to the original will allow the comparison of the percentiles and their relative changes, as well as evaluate the overall impact of performing the augmentation. Transparency would also be fostered if any computer code presented is accompanied by comments that can lead the reader through the paradigm with sufficient data to allow for duplication of the process. A concise description of the rationale for the sequence of calculations and how they may be interpreted could be included in the comments or as a separate section.

2.9 Summary

The MCA and the MEE included in the HHRA fall short of what can and should be done to evaluate risks due to fish and waterfowl consumption. Those models do not make full use of the available data distributions, artificially manipulate existing data, and are not designed to capture inter-dependencies among the individual parameters. In consequence, the results tend to reproduce the simpler analyses rather than improve upon them. The increasing complexity of each tier of the model should not produce the same results but should provide a refined approximation of actual risks (EPA, 2001). A more robust MEE model that incorporates actual data distributions, considers inter-dependencies among the data distributions, allows for correlations, and addresses the other concerns discussed above would provide a final risk distribution that is more representative of the population of interest and ultimately of more utility to risk managers.

3.0 Alternative MEE Analysis of Fish Consumption

To quantify the impact of the issues discussed above, AMEC has performed an alternative MEE analysis of potential risks due to the consumption of fish from each reach of the Housatonic River. A full report on this analysis is provided in Exhibit H.1 to this Attachment. This analysis, which evaluates risks from total PCBs (tPCBs), demonstrates the differences in results obtained when the actual data are used in the MEE (instead of expanded statistical summaries of the data), when appropriate inter-dependencies among variables are considered, and when the other concerns discussed above are addressed.

3.1 Methods

This alternative MEE analysis incorporates the following changes in exposure parameter distributions and inter-dependencies of parameters, in accordance with the above discussion:

- It uses all the raw tissue tPCB data available from each reach of the river by species to develop a distribution of EPCs for each reach and species.

- For the fish consumption rate distributions, it uses the data from the Ebert et al. (1993) angler survey as reported by the study's authors and provided electronically to EPA. The data for river/stream fish consumption rates have been applied to Reaches 5-6 and 11-12 and the data for lakes/ponds have been applied to Reaches 8 and 14-15. Consistent with the data provided in the survey, the consumption rates reflect sharing among household members as reported by the survey respondents.

- It does not include any artificial expansion of the fish consumption distributions to include hypothetical upper bounds outside of the actual distributions.

- It considers the inter-dependencies among input parameters as discussed above.

- It expands cooking loss factors to include all cooking methods reported in the Ebert et al. (1993) angler survey and uses published cooking loss studies for low lipid level fish (in combination with the data used by EPA) to derive cooking loss factors for those cooking methods.

- It uses age- and gender-specific Berkshire County census data on mortality and mobility to develop a distribution of exposure durations for the Massachusetts reaches. For the Connecticut reaches, age- and gender-specific census data on mortality in Litchfield, Fairfield, and New Haven Counties, combined, and mobility data for Berkshire County were used to develop a distribution of exposure durations.

The MEE model was run first with the above changes in the exposure parameter inputs, but using point estimates for the Cancer Slope Factor (CSF) and the RfD for PCBs. In this model run (MEE 1), the upper bound CSF of 2 $(mg/kg\text{-}day)^{-1}$ recommended by EPA (1996, 2003) was

used to evaluate carcinogenic risks. For non-cancer hazards, EPA's chronic RfD of 2E-05 mg/kg-day (EPA, 2003) was used to evaluate all exposures that were seven years or more in duration, while, consistent with EPA (1989) guidance, exposures that were less than seven years in duration were evaluated with EPA's subchronic RfD of 5E-05 mg/kg-day (EPA, 1997b).

The model was then run a second time (MEE 2) with the same exposure input parameters, but using distributions of the toxicity values in place of point estimates, to assess the additional uncertainties associated with those values. These distributions and their bases are described in detail in Exhibit H.2. Specifically, the distribution of RfDs was derived by using the same equation as that used by EPA to derive its RfD for Aroclor 1254, substituting distributions for the point-estimate uncertainty factors used by EPA, and then using a probabilistic technique (Monte Carlo Analysis with Latin Hypercube) to determine the uncertainty in the estimate of the population threshold. To develop the input distribution for the CSF, the central and upper-bound slope estimates derived in the same studies considered by EPA (1996) in its reevaluation of the cancer potency of PCBs were used (Kimbrough et al., 1975; NCI, 1978; Schaeffer et al. 1984; and Norback and Weltman, 1985; Brunner et al., 1996). Because the PCBs found in the Housatonic River and its floodplain most closely resemble Aroclors 1260 and 1254, only the data provided in those studies for these two Aroclors were included in the development of the CSF distribution. In addition, because female rats appear to be more sensitive to PCBs than male rates, only the female rat data were used.

3.2 Results

The alternative MEE results for the fish consumption pathway for each river reach and each model run are summarized in Table 1. Table 1 also provides comparisons of the cancer risks and non-cancer hazards that were predicted by AMEC using its alternative MEE analyses with those predicted in the HHRA. The results of these alternative analyses indicate that cancer risk and non-cancer hazard estimates are substantially lower than the risk and hazard estimates presented in the HHRA for the corresponding reaches when the MEE is refined to accommodate the full range of data, inter-variable correlations, and the other modifications identified above.

Table 1. Comparison of tPCB Cancer and Non-Cancer Risk Estimates from EPA's MEE Analysis with Those from Alternative MEE Analyses Conducted by AMEC

	Cancer Risks					
	Adult/Child					
	50th%ile			95th%ile		
	EPA HHRA	AMEC MEE		EPA HHRA	AMEC MEE	
River Reach	MEE	Alternative MEE 1[a]	Alternative MEE 2[b]	MEE	Alternative MEE 1[a]	Alternative MEE 2[b]
5 to 6	2E-03	5E-05	2E-05	8E-03	8E-04	4E-04
8	2E-03	4E-05	1E-05	6E-03	6E-04	2E-04
11 to 12 (trout)	3E-04	9E-06	3E-06	1E-03	2E-04	6E-05
11 to 12 (bass)	2E-04	5E-06	2E-06	7E-04	8E-05	3E-05
14 to 15	1E-04	5E-06	2E-06	5E-04	8E-05	3E-05

	Non-cancer Hazard Indices											
	Adult						Child					
	50th%ile			95th%ile			50th%ile			95th%ile		
	EPA HHRA	AMEC MEE		EPA HHRA	AMEC MEE		EPA HHRA	AMEC MEE		EPA HHRA	AMEC MEE	
River Reach	MEE	Alternative MEE 1[a]	Alternative MEE 2[b]	MEE	Alternative MEE 1[a]	Alternative MEE 2[b]	MEE	Alternative MEE 1[a]	Alternative MEE 2[b]	MEE	Alternative MEE 1[a]	Alternative MEE 2[b]
5 to 6	40	5.4	0.42	500	69	7.4	91	3.1	0.33	1000	49	6.6
8	29	4.3	0.33	330	44	4.6	61	2.7	0.28	720	28	3.9
11 to 12 (trout)	3.7	0.95	0.07	64	12	1.3	7.9	0.65	0.07	110	7.8	1.1
11 to 12 (bass)	3.7	0.50	0.04	44	6.2	0.65	7.7	0.36	0.04	91	4.0	0.57
14 to 15	2.4	0.56	0.04	31	5.8	0.60	5.3	0.35	0.04	61	3.6	0.50

[a] Alternative MEE 1 conducted using point estimate toxicity values

[b] Alternative MEE 2 conducted using a distribution of toxicity values

For MEE 1, which used point estimate toxicity values, the cancer risk estimates at the 50th percentile are slightly more than an order of magnitude lower than EPA's estimates, while estimates at the 95th percentile are slightly less than an order of magnitude lower. This is to be expected due to the fact that EPA's reduction of some parameter distributions to a summary statistic (e.g., use of the single 95% UCL concentration as the EPC) tends to collapse the distributions toward average values rather than allowing the distinctive characteristics of the data themselves to be expressed. Thus, in this alternative MEE, the output distribution is not as "flat" as in EPA's MEE in that the central tendency estimates are lower but the tails of the distribution are greater. This is a more representative output as one would expect that higher exposure levels, which are the result of more extreme behaviors, would have a low probability of occurrence.

For non-cancer hazards, the 50th and 95th percentile Hazard Indices (HIs) for adults in MME 1 are somewhat less than an order of magnitude lower than in the HHRA. For children, the predicted HIs are substantially lower in MEE 1 than in the HHRA.

As discussed in more detail in Exhibit H.1, the lower risk and hazard estimates derived in the alternative MEE 1 analysis are likely to be largely the result of two changes in the modeling approach: (1) the direct use of the fish consumption rate distributions reported by Ebert et al. (1993) for rivers/streams or lakes/ponds, as appropriate, including sharing among household members, without artificial expansion of the upper bounds; and (2) the use of the discrete fish sampling data as an input distribution rather than the upper bound point estimate used by EPA.

Even greater differences are observed between the results reported in the HHRA and those predicted using the MEE 2 model. CTE cancer risks using the CSF distribution are lower than those predicted in the HHRA by roughly two orders of magnitude, while the 95th percentile cancer risks are lower by more than an order of magnitude. Use of a distribution of RfDs yields even more striking reductions in the non-cancer HIs predicted in the HHRA. For adults, the HIs in the MEE 2 model are generally about two orders of magnitude lower than those predicted in the HHRA for both the 50th and the 95th percentile estimates, while the HIs for children are generally reduced by well over two orders of magnitude compared to the HIs in the HHRA. The differences result from a combination of the factors discussed above for MEE 1 and the additional consideration of the uncertainties associated with the dose-response values.

4.0 Summary and Conclusions

For the reasons discussed above, GE believes that the MEE in the HHRA should be modified to reflect the types of changes in exposure parameter inputs that are illustrated for the fish consumption pathway in AMEC's alternative MEE 1. By avoiding the use of summarized data and conjectural upper bound estimates and incorporating correlations among input parameters, such a model will be more reflective of the underlying data and of the behaviors of the potentially exposed population.

In addition, GE submits that the MEE model would be further improved by including, at least as a sensitivity analysis, an additional quantitative evaluation of the uncertainties in the dose-response values, as illustrated in AMEC's alternative MEE 2.

While GE has not provided an alternative MEE analysis for the waterfowl consumption scenario, it recommends that EPA also revise the MEE analysis for that scenario in the same way. This will ensure that waterfowl risks and hazards are as representative of potential exposures and risks as they can be.

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EXHIBIT H.1

ALTERNATIVE MICROEXPOSURE EVENT (MEE) ANALYSIS OF FISH CONSUMPTION FOR THE REST OF RIVER

EXHIBIT H.1

Alternative Microexposure Event (MEE) Analysis

of Fish Consumption for the Rest of River

On behalf of General Electric (GE), AMEC has conducted an alternative Microexposure Event (MEE) analysis to quantify potential health risks associated with PCBs to fish consumers along the Housatonic River. AMEC has separately evaluated Reaches 5-6 (confluence to Woods Pond Dam), Reach 8 (Rising Pond), Reaches 11-12 (West Cornwall and Bulls Bridge) and Reaches 14-15 (Lakes Lillinonah and Zoar) using total PCB data. For each run, cancer risks were estimated for combined childhood and adult exposures. Non-cancer hazards were estimated separately for children (ages 1-6 years) and adults. For the Cornwall area, separate estimates were derived for trout fishing and for bass fishing, to be consistent with the approach used in the HHRA.

Overview of MEE Analysis

As is the case with any probabilistic risk assessment, MEE analysis provides an estimate of the likelihood or probability of risk associated with the entire range of exposure. The probability of risk and range of exposure are estimated by substituting point estimate values with values from probability distributions. However, MEE analysis not only estimates risks associated with the range of exposure, but it also allows one to model variation in exposures over time by aggregating and summing independent exposure events over an individual's lifetime. In this way, it can capture changes in individual behaviors and conditions over time (Simon, 1999). The theory and detailed methodology behind MEE analysis are documented in the literature (Harrington et al., 1995; Price et al., 1996; Keenan et al., 1996a; Simon, 1999) and in EPA Guidance (EPA, 2001). In brief, an individual's total exposure to a constituent is calculated by summing the doses received during many individual exposure events. Each event is simulated using information specific to the time and location of the exposure event. The number of events and sequence in which they occur in the person's life can be simulated based upon information about individuals' short- and long-term behaviors.

Modeling long-term exposures as a summary of separate events is not new; in fact, this approach was recommended by EPA (1992) for evaluating exposures that occur primarily during childhood, when body weights change rapidly. MEE analysis has been used by EPA and

by independent researchers to simulate duration of residential exposure (Johnson and Capel, 1992; Sielken, 1994). It also has been used to evaluate childhood exposures to lead (Goodrum et al., 1994), exposure to contaminants in tap water (Harrington et al., 1995), and exposure to dioxins from the consumption of freshwater fish (Keenan et al., 1993a,b; 1995; 1996b; 1997a,b). MEE analysis was employed in the supplemental risk assessment for the Stringfellow Superfund site in California (Pyrite Canyon Group, 1994; Keenan et al., 1996a). Recently, MEE analysis has been described in EPA's *Risk Assessment Guidance for Superfund: Volume 3, Part A -- Process for Conducting Probabilistic Risk Assessment* as a viable alternative for modeling time-dependent variability in concentrations, daily activity patterns, and other behavioral exposure factors (EPA, 2001). In essence, the use of MEE analysis can more effectively characterize the impact of variability or uncertainty in input parameters on the estimates of dose rates in an exposed population by considering time-dependent changes.

Central to the MEE modeling approach is the shift in emphasis from generalizations about a whole population's exposure to application of relevant information to estimate the range of exposures for individuals within the exposed population. Under this approach, an individual's exposure is viewed as a series of separate events and the exposure received from each event is modeled independently. Taken together, these discrete exposure events create an exposure history for each individual within the exposed population.

This MEE analysis evaluates exposures resulting from each fish meal consumed by each angler. (In this Attachment, the term "angler" is used as a shorthand to refer to any consumer of sport-caught fish from the Housatonic River.) As depicted in Figure 1, the model determines the age, body weight, and fish consumption rate of each angler during each year of exposure, as well as the angler's exposure duration. The model also determines the characteristics of each fish meal. These characteristics include the fish species consumed, the PCB concentration in the fish, the method used to prepare the fish, and the level of PCB loss resulting from the cooking practices used. In the end, the total dose received by each angler is the sum of the discrete exposures received by that angler as a result of all of the specific fish meals consumed. Using this approach, correlations between exposure parameters can be easily modeled and limitations in data sets can be incorporated into the model.

MEE defines the Lifetime Average Daily Dose (LADD) for fish consumption as the sum of potential short-term (e.g., daily, annual) exposures represented by the following equation:

$$LADD = \frac{1}{LT} \sum_{j=1}^{Angling\ Duration} \frac{1}{BWj} \sum_{i=1}^{FishMeals\ Eaten} ((FishConc._{ij})(1 - CookingLoss_{ij}))$$

where,

Angling Duration	=	the period of time in years that an angler fishes the river
Fish meals Eaten	=	the number of fish meals consumed in the jth year
Fish Conc.$_{ij}$	=	the PCB concentration in the ith fish caught in the jth year
Cooking Loss$_{ij}$	=	the fraction of PCBs lost during the cooking of the ith fish caught in the jth year
BW$_j$	=	the average weight of the individual during the jth year of his/her life
LT	=	a standard lifetime for humans

Structure of the MEE Model

MEE allows the incorporation of age-related exposure factors into the estimates of long-term dose rates by adjusting angler- and age-specific parameters (e.g., body weight) for each year of exposure. In addition, by modeling each fish meal separately, MEE analysis considers the varying species consumed, concentrations in those species, and the cooking methods use to prepare them. Finally, the duration of an individual angler's exposure is characterized, not by adoption of an independent distribution of durations, but by using information on the angler's age at the time the exposure begins, together with age-specific rates of mobility and mortality, to predict the length of exposure.

The MEE model processes the following steps in calculating potential exposures and risks due to PCBs in fish consumed:

1. The number of anglers to be evaluated is entered. For this model, a total of 50,000 anglers has been selected to ensure stability of the output.

2. The first angler is selected. His/her specific characteristics are selected at random from the appropriate probability distributions (discussed below), including age at the initiation of the model (start age), a fish consumption rate percentile, and a body weight percentile. Assigning the same percentile of the fish consumption rate distribution to that individual throughout his/her exposure period incorporates a temporal correlation for fish consumption

rate. Thus, during each year of exposure, the same fish consumption rate is used for that individual to provide consistency in behavior from year to year. Similarly, a correlation for body weight is incorporated in the model by assigning that individual to a specific percentile of the body weight distribution throughout the exposure period. For example, an individual assigned a 25th percentile body weight at the start of the model will continue to have a 25th percentile body weight throughout the exposure duration. Consequently, weight will change on an age-specific basis, because the body weight distributions change with age, but the individual will always be at the 25th percentile for body weight.

3. The first year of exposure is initiated.

4. A specific body weight is selected based on the percentile selected initially and the angler's age during the first year.

5. A daily probability of consuming a fish meal is derived based on the selected fish consumption rate.

6. The first day of the year is selected. Whether or not the angler consumes a fish meal that day is determined by random draw based on the daily probability of consuming a fish meal.

7. If the angler consumes a fish meal, a species is selected along with a fish tissue concentration.

8. Based on the species consumed, a cooking method is selected from the probabilities for species-specific cooking methods and the appropriate cooking loss factor is applied. PCB intake from that meal is calculated and saved for that angler.

9. Fish consumption on subsequent days of the first year is calculated in the same way until 365 days have been evaluated for the year.

10. At the end of the year, the model identifies whether the angler dies or moves away, based on mobility and mortality demographic data. If the angler does not die or move away, his/her exposure is evaluated for a second year and the dose for the second year is added to the dose for the first year. These annual iterations continue until the angler either dies or moves away from the counties along the Housatonic River.

11. Once all years of exposure have been evaluated for the individual angler, doses are summed and averaged, over the total number of years of exposure for noncancer hazards, and averaged over a lifetime (75 years) for cancer risks. The cancer slope factor (CSF) is applied to the estimated cancer dose to derive a cancer risk estimate; the non-cancer dose is divided by the reference dose (RfD) to derive a hazard index (HI). The estimated cancer risk and non-cancer HI for that individual angler are saved.

12. The model then goes on to evaluate the next angler until the total number of anglers specified at the start of the model run (50,000) has been met.

13. Once all anglers have been evaluated, summary statistics for cancer risks and non-cancer hazards for the entire population of modeled anglers are calculated and presented as a full distribution of risk and hazard results.

Model Inputs – Exposure Parameters

Sources of data and types of input distributions for each exposure parameter are discussed in the following sections. Cooking preference, cooking loss, and body weight input distributions were the same for each model run, while fish consumption rates, fish tissue concentrations, and species preference were reach-specific. Exposure duration estimates were derived from census data for Berkshire County in Massachusetts and for Litchfield, New Haven, and Fairfield Counties in Connecticut.

Start Age

To be consistent with EPA's approach, the MEE analysis evaluated exposures to individuals between the ages of one and 90 years who may eat fish from the Housatonic River. As discussed previously, when the model is initiated for each angler, it begins by selecting a "start age" for that individual. The start age for that individual is the age at which the hypothetical angler begins to consume fish from the Housatonic River. A number of factors affect the start age within the real population. Individuals who grow up in the area and whose parents like to fish the river may begin to eat fish early during their childhood. Others may not begin to eat freshwater fish until they acquire a taste for it during adolescence. Still others may not begin to eat fish from the Housatonic River until adulthood, particularly if they did not move to Berkshire County until they were adults. Thus, individuals could begin eating fish from the river at any

time during their lives. For this model, the distribution of "start ages" is intended to capture the range of ages at which anglers begin to consume fish from the Housatonic River.

In this MEE, it was possible to begin eating fish from the Housatonic River at any age, beginning at 1 year of age. The probability of starting to eat fish at each year of age was based on population statistics for each age group. To develop a distribution of start ages for Massachusetts' anglers, 2000 census data for Berkshire County were used. Start ages for Connecticut anglers were based on the combined 2000 census data for Litchfield, Fairfield and New Haven Counties. Age- and gender-specific population statistics for these Connecticut counties were combined to estimate the total population size of each reported age grouping. Gender-specific census data for the following age categories were used: 0 to 4, 5 to 9, 10 to 14, 15 to 19, 20 to 24, 25 to 29, 30 to 34, 35 to 39, 40 to 44, 45 to 49, 50 to 54, 55 to 59, 60 to 64, 65 to 69, 70 to 74, 75 to 79, 80 to 84 and 85+ years. The population for each age category was divided by the total population, and then divided by the number of years included in the category to derive an estimated probability for each year of age. For example, the total three Connecticut county population of males for the 20 to 24 year old category was divided by the total population of males of all ages in those three counties and then by five (5) to estimate the size of the population at each discrete age (i.e., 20, 21, 22, 23 and 24 years). It was then assumed that there was an equal probability that an angler would start fishing at any of those ages.

To ensure that at least 10 years of exposure were possible for each angler modeled in the evaluation of potential cancer risks, the start age distribution was truncated at age 80. This ensured that every hypothetical angler whose exposure was modeled had the potential to be exposed for at least 10 years, since the model was allowed to run for a maximum of 90 years. Start ages for the 1 to 6 year old child were derived in a similar fashion, but the distribution was truncated to age 6. Cumulative start age probabilities are shown in Table 1.

Fish Consumption Rates

Recreational anglers who catch and consume fish from the Housatonic River have the potential to be exposed to PCBs that have accumulated in the fish tissues. It is, therefore, important to attempt to capture the consumption behavior of those individuals so that potential risks due to fish consumption can be more accurately assessed.

The raw data from the Maine angler survey conducted by Ebert et al. (1993) were used as the basis for the fish consumption rate input distributions. For the reasons discussed in Attachment G to this set of comments, the following consumption rate data were used: For Reaches 5-6 and 11-12, the fish consumption rates for "consuming anglers" for "rivers and streams" (i.e., those Maine angler survey respondents who reported that they consumed at least one fish meal caught in a river or stream in Maine) were used. For Reach 8 and Reaches 14-15, the fish consumption rates for "consuming anglers" for "lakes and ponds" were used. The input distributions for adult fish consumption rates are summarized in Table 2. Fish consumption rates for young children (aged 1 to 6 years) were conservatively assumed to be 40 percent of the adult rates based on data on freshwater finfish consumption provided by Rupp et al. (1980).

For each angler modeled in the MEE analysis, an initial annual consumption rate was selected randomly from the distribution of possible rates. This annualized consumption rate was then converted to a daily probability of consumption and the model estimated exposure for the first year. This daily probability was then used for every subsequent year of exposure. This approach of restricting fish consumption rates to a fixed percentile throughout an individual's exposure duration follows that suggested by EPA's probabilistic risk assessment guidance as a viable means for modeling time-dependent variability in behavioral exposure factors (EPA, 2001).

Fish Tissue Concentrations

Tissue concentrations of Housatonic fish that are consumed are variable and depend on the species consumed and the river reach being evaluated. All of the fish tissue data used by EPA to derive its exposure point concentration for fish tissue (Tables C.3-1 to C.3-6 of the HHRA) were input to the MEE model by species and by reach, allowing an equally weighted probability that any one reach- and species-specific fish tissue concentration would be selected for each fish meal.

For each fish meal consumed by an angler in this MEE analysis, the species of the fish was first selected based on the species preference probability distribution for the reach being evaluated (discussed in the following section). Once a fish species was selected for a meal, the fish tissue concentration was randomly selected from the available concentration data for that species in that river reach. Only total PCB concentration data were used in the analysis.

Species Preference

Anglers are likely to catch and consume certain species of fish more frequently than other species. Contaminant levels in the tissue of Housatonic River fish vary among species, with the highest concentrations in bass and bullhead and the lowest concentrations in perch and sunfish. While anglers are likely to catch and consume certain species of fish more frequently than other species, and it would be optimal if data were available to capture this variation in a species preference probability distribution, such data were not available for the Housatonic River fishery. Thus, in order to represent the full data set, AMEC assumed an equal preference for catching and consuming each species in each reach for which there were sampling data. Thus, for Reaches 5-6 and Reach 8, the probability of catching and consuming bass, yellow perch, bullhead, and sunfish were each 25 percent. For Reaches 11-12, for which separate analyses were done for trout and smallmouth bass, the probability of selecting each species in the relevant analysis was 100 percent. Similarly, since there are only data available for smallmouth bass in Reaches 14 and 15, the probability of catching and consuming smallmouth bass was assumed to be 100 percent.

Cooking Method

The cooking method used to prepare fish can result in a reduction of PCB mass prior to consuming the fish. The appropriate input parameter for the risk assessment represents that amount of chemical actually ingested, rather than the amount originally present in the fish. The magnitude of the reduction of chemical mass due to cooking is dependent on the cooking method used. The reduction in chemical mass is expressed as the fraction of the chemical mass that remains in the cooked fish relative to the uncooked fish.

Certain types of fish tend to be cooked in certain ways. For the MEE analysis, species-specific cooking preferences were developed, based on the Maine angler survey data from the Ebert et al. (1993) study. In that survey, respondents were asked to list their most preferred species for consumption and to indicate the way in which they usually cooked them. A total of seven cooking methods were provided as options for response. These were raw, baked, broiled/grilled, fried, poached, boiled, and soup/stew/chowder. AMEC combined the data for poaching, boiling and making soup into a single category, resulting in five possible species-specific cooking methods. Based on these data, the probabilities of using each of these methods to cook the bass, bullhead, perch, sunfish, and trout were developed and are provided

in Table 3. After the model selected the species of each fish meal, the likely method of cooking was selected at random from the cooking method probabilities for that species.

Cooking Loss Factors

Different cooking methods result in a range of reductions of PCB levels in fish tissues. In its evaluation of cooking loss factors for PCBs, EPA considered only those cooking loss studies of fish with lower lipid levels. This is appropriate given the low lipid levels in most Housatonic River fish. The studies upon which EPA's cooking loss factors were based included studies of lake trout (Daubenmire, 1996; Zabik 1996; Wang and Harrad, 2000) and white bass (Daubenmire, 1996). Because these authors only evaluated broiling, salt boiling, and frying, these were the only cooking methods considered by EPA. There are additional studies, however, that evaluated PCB losses after frying and baking fish with low lipid levels (Smith, 1972; Smith et al., 1973; Skea et al., 1981; and Puffer and Gossett, 1983). While EPA did not consider these studies, they are also relevant and thus were included in AMEC's evaluation of cooking loss factors (Table 4). Because fish consumers may cook the fish with or without the skin, the cooking loss factors for skin-on and skin-off fillets have been evaluated together, assuming that for some species the fish will be cooked with the skin on while for others the skin will be removed.

Based on AMEC's evaluation of these data, the percent of PCBs lost due to broiling or grilling ranges from 7 to 25 percent with an average of 18 percent. (This is identical to the cooking loss assumption used in the HHRA for this cooking method.) The percent of PCBs lost after baking range from 10 to 16 percent, with an average of 13 percent. For frying, the cooking losses range from 17 to 74 percent with an average of 37 percent. For boiling (poaching, boiling or making soup), the cooking loss ranges from 10 to 13 percent with an average of 12 percent. These average reductions in PCB levels in fish tissue were used to estimate PCB losses when the corresponding cooking method was selected in the MEE model (Table 5). Because there were a few respondents to the Maine angler survey who reported that they had not cooked certain fish that they consumed, "raw" was also provided as a species-specific cooking option in the MEE model. When the model selected "raw" as the cooking method, it was assumed that there was no PCB loss (Table 5).

Body Weight

The variability associated with body weights was also characterized in this model. Two primary sources referenced in the *Exposure Factors Handbook* (EPA, 1997a) were used to generate body weight percentile distributions. The first study (Burmaster et al., 1994) reported body weight distributions for children between the ages of 6 months and 19 years. Body weight distributions for adults ages 18 to 74 were reported by Brainard and Burmaster (1992). The percentile distribution for ages 74 to 90 were assumed to be equivalent, as no data were available above age 74 and little variation is expected in body weight after this age. The gender-specific body weight probabilities incorporated into the model are presented in Tables 6 and 7 for females and males, respectively.

While the MEE model accounts for temporal variability in body weight, an individual was assumed to remain within the same body weight percentile throughout his/her exposure duration so that body weight changes proportionally over time. For example, if an individual was assigned to the 25th body weight percentile at the start of the model, that individual was assumed to remain at the 25th percentile for the remainder of his/her exposure.

Exposure Duration

For the MEE analysis, the exposure duration is a function of the probability that each individual angler evaluated continues to fish the Housatonic River on subsequent years. There are a number of factors that affect the likelihood that an angler continues to fish from year to year. Some anglers may fish sporadically over a long period of time. This fluctuation in fishing activity may be a result of schedule changes, illness, or lack of strong interest in fishing regularly. At present, however, there are no reliable long-term data that allow one to predict with adequate certainty the potential for an angler to fish during each subsequent year based on these time-variable factors.

There are two factors, however, which directly impact the ability of an angler to fish the area from year to year and which can be evaluated based on available data. The first is the probability that an individual angler may die in a given year and thus will not be able to continue to fish during subsequent years. The second is that anglers may move out of the area during the exposure period. If they move far enough away, it is unlikely that they will continue to fish along the Housatonic River.

The MEE model evaluates exposure duration as a two-stage process. Once an angler of a specific age has been selected and his/her exposure estimated for the first year, the model cycles through to the next year of age and asks whether the angler dies during that year. The probability that the angler will die is based on local demographic data related to mortality rates for each age group. Logically, the probability that an angler will die is very small for young anglers but increases as the age of the angler increases. If the angler does not die during a given year, the model asks whether the angler moves out of the area during the next year. Census data on population mobility for given age groups have been used to derive the estimated probabilities that anglers of a certain age will move away from the exposure area. If the angler does not move during that year, the model evaluates exposure for the subsequent one-year period and continues to cycle in this fashion until the angler either dies or moves away. If the angler moves or dies at the end of that year, the exposure is terminated and total exposure over all years of exposure is summed.

Berkshire County surrounds the Housatonic River in Reaches 5-6 and 8 in Massachusetts. In Connecticut, Fairfield, Litchfield and New Haven Counties border the river. It is assumed that the majority of anglers who fish the Housatonic River regularly live reasonably close to it and thus live in one of these four counties. Accordingly, these four counties were used as the population "study area" for review of mortality statistics and mobility data. The derivations of the mortality and mobility probability distributions are discussed below.

Mortality

The probability that an individual angler may die during a specific year of his or her life affects the number of years that the individual can be expected to catch and consume fish from the river. Standard actuarial mortality tables can be used to predict the life expectancy of a given angler and to determine whether an angler of a specific age is likely to remain a member of the angler population during each modeled year of exposure.

Mortality data for Berkshire County were available from the Massachusetts Department of Public Health for the year 2001. These data were provided as total deaths by age and gender. Mortality data for the three Connecticut counties were available from the Connecticut Department of Public Health (personal communication, Charles Nathan, CDPH) for the year 1997.

The census data age groupings used in the MEE analysis were: ages 0-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, and 85 and older. For Berkshire County, the number of deaths reported for each age group for each gender was divided by the total county population size for that age group/gender to derive a gender- and age group-specific death rate. For example, according to MDPH data, a total of 12 Berkshire County males between the ages of 40 and 44 years died in 2001. According to 2000 census statistics, a total of 5,161 males between these ages lived in Berkshire County. This means that, over the male population of 40 to 44 year olds, the probability of dying was 0.002325 (12/5161). Thus a probability of dying of 0.002325 was assigned to each year of age in this age group. With the exception of summing the numbers of age group-specific deaths for the three Connecticut counties and dividing by the combined three-county population sizes for those age groups, mortality probabilities for the three combined Connecticut counties were similarly derived.

Table 8 shows the gender- and age-specific mortality rates for Berkshire County, and Table 9 reports the rates for the three Connecticut county area. These values were incorporated into the MEE model.

Mobility

Just as mortality affects the exposure duration for the risk assessment, so does the mobility of the angler population (i.e., its tendency to move away). Many individuals can be expected to move from one residence to another at least once during their lifetimes. In some cases, anglers who currently live in one of the counties proximate to the Housatonic River may move to new residences that are substantially removed from the area. It is likely that, under those circumstances, such individuals will stop fishing the Housatonic River and instead choose to fish at alternative fisheries located closer to their new homes. Thus, in developing a realistic exposure model, it is important to consider the effect that age-specific mobility has on the cessation of angling the Housatonic River.

To evaluate this parameter, county-specific census data on population mobility can be evaluated to determine the probability that an individual would move far enough from the area that his or her angling activity in the Housatonic River would cease. Many individuals reported as movers in the census data changed residences within Berkshire County but did not leave the

County. It was assumed that these individuals would continue to fish the Housatonic River despite these moves. Thus only those individuals who moved out of Berkshire County were considered “movers” for this analysis.

Unfortunately, Massachusetts and Connecticut, unlike other states, have not compiled or were not willing to share their mobility information on a county-specific basis. In order to derive county-specific in-migration and out-migration statistics, AMEC compiled the data on a town-by-town basis in Berkshire County. These data were used as the basis for determining mobility rates for Massachusetts and Connecticut. We believe that these mobility rates are a reasonable surrogate for northern Connecticut, which is rural and similar to Berkshire County outside of Pittsfield, but very conservative (lower frequency of moving) for the more urban towns further south in Connecticut because census data generally indicate that individuals who live in rural areas move less frequently than individuals who live in more urban areas (EPA, 1997a; Tables 15-163 and 15-164). In addition, as discussed below, the exposure duration distribution generated using mortality and mobility census data was similar to the exposure duration distribution, based on the MADPH survey data for residence time, that was used in EPA’s HHRA.

Population mobility statistics for Berkshire County were used to develop the probability distributions for cessation of angling for each age group. To estimate the probability that an individual of a specified age would leave the area, the number of "out-migrants" from Berkshire County between 1985 and 1990 was divided by the sum of the populations of "non-movers" and "out-migrants" using the following equation:

5 year probability = out-migrants/(non-movers + out-migrants)

This resulted in a probability of moving out of Berkshire County over a five-year period (between 1985 and 1990). This 5-year probability was then adjusted to correct for the probability of moving out of the area that occurred during each of the five years (assuming that the probability is equivalent for each of the years), using the following equation:

$$M_1 = 1- (1-M_5)^{1/5}$$

where,

M_1 =probability of moving in any one year

M_5 =probability of moving in five years

Simply dividing the 5-year probability by five does not determine the relationship between the probability of moving in one year and the probability of moving in five years. This is because once some fraction of a population has moved in the first year, they are no longer available to move in subsequent years.

One-year mobility probabilities for each year of age are presented in Table 8 for Berkshire County. Probabilities of moving away from the study area in any single year ranged from a low of 0.00258, for individuals aged 46 to 49, to a high of 0.179 for individuals 85 and older. As discussed previously, the data on population mobility for Berkshire County were also applied to the tri-County area in Connecticut for the MEE of the Connecticut reaches of the river.

Averaging Time

The averaging time used for the assessment of carcinogenic risks was 75 years, the value recommended by EPA (1997a) to represent lifetime exposure. For the non-cancer hazard assessment, the averaging time was equal to the exposure duration. Thus, if the model predicted that an individual angler fished for 30 years, the non-cancer averaging time was also assumed to be 30 years.

Model Inputs – Toxicity Values

As the last step in evaluating each angler in the MEE model, the potential carcinogenic and non-carcinogenic health risks are quantified by comparing the estimated doses with the appropriate CSF and RfD, respectively. Lifetime incremental cancer risk for the angler is calculated as the product of the LADD and the CSF. Non-carcinogenic chronic risks are evaluated by dividing an average exposure level (average daily dose or ADD) corresponding to a chronic exposure duration by the chronic RfD to derive the HI. For individuals who have a subchronic exposure duration (less than seven years of exposure, as defined by EPA, 1989), the subchronic HI is calculated in the same way except that the subchronic RfD is used as the dose metric.

Cancer risks and non-cancer hazards were calculated in two different ways in this MEE analysis. The first alternative MEE analysis (MEE 1) used point estimate toxicity values derived by EPA. To calculate cancer risks, the upper bound point estimate CSF of 2 $(mg/kg\text{-}day)^{-1}$ for PCBs, recommended by EPA (1996) and published in EPA's IRIS (EPA, 2003) database, was used, as in the HHRA. For non-cancer hazards, EPA's chronic RfD of 2E-05 mg/kg-day for Aroclor 1254, as recommended in IRIS (EPA, 2003) and used in the HHRA, was used to evaluate all chronic exposures, while subchronic exposures were evaluated using the subchronic RfD of 5E-05 mg/kg-day for Aroclor 1254 recommended by EPA (1997b).

The second alternative MEE analysis (MEE 2) used a distribution of CSF and RfD values to capture the additional uncertainty associated with these dose metrics. The methods used to derive these distributions are discussed in detail in Exhibit H.2 of these comments. In brief, the CSF distribution was developed utilizing the central and upper bound slope estimates for female rats that were reported in the same studies of Aroclors 1254 and 1260 that were considered by EPA (1996) when it developed its point estimate CSF. That input distribution is summarized in Table 10. For non-cancer hazards, the distribution of chronic RfDs was derived using the method presented by Swartout et al. (1998). This method involved replacing the point estimate uncertainty factors used by EPA (2003) to derive its upper bound RfD for Aroclor 1254 with distributions of those uncertainty factors and then using a probabilistic model to generate the range of RfDs and their associated probabilities. The distribution of subchronic RfDs was developed using the same method except that the uncertainty factor used by EPA (2003) to account for estimating chronic toxicity based on subchronic exposure was not included. The resulting distributions of RfDs are also summarized in Table 10.

Results

Both alternative MEE analyses separately evaluated non-cancer risks to young children, ages 1 to 6 years, and to older children and adults, ages 7 and older. Cancer risks were evaluated over a lifetime of 75 years. The outputs of the MEE analyses exist in the form of distributions of predicted cancer risks and non-cancer HIs.

The estimated 50^{th} and 95^{th} percentile cancer risks from the two alternative MEE analyses of the fish consumption pathway for each river reach are as follows:

	Lifetime Cancer Risks Adult/Child			
	Alternative MEE 1		Alternative MEE 2	
River Reach	50th %ile	95th %ile	50th %ile	95th %ile
5 to 6	5E-05	8E-04	2E-05	4E-04
8	4E-05	6E-04	1E-05	2E-04
11 to 12 (trout)	9E-06	2E-04	3E-06	6E-05
11 to 12 (bass)	5E-06	8E-05	2E-06	3E-05
14 to 15	5E-06	8E-05	2E-06	3E-05

The estimated 50th and 95th percentile non-cancer HIs from the two MEE analyses are as follows:

	Noncancer Hazard Index				Noncancer Hazard Index			
	Alternative MEE 1		Alternative MEE 2		Alternative MEE 1		Alternative MEE 2	
	Adult		Adult		1-6 Year Child		1-6 Year Child	
River Reach	50th %ile	95th %ile	50th %ile	95th %ile	50th %ile	95th %ile	50th %ile	95th %ile
5 to 6	5.4	69	0.42	7.4	3.1	49	0.33	6.6
8	4.3	44	0.33	4.6	2.7	28	0.28	3.9
11 to 12 (trout)	0.95	12	0.07	1.3	0.65	7.8	0.07	1.1
11 to 12 (bass)	0.50	6.2	0.04	0.65	0.36	4.0	0.04	0.57
14 to 15	0.56	5.8	0.04	0.60	0.35	3.6	0.04	0.50

Discussion of Uncertainties

An important facet of human health risk assessment concerns the recognition of uncertainties and limitations inherent in the process. Uncertainties specific to these alternative MEEs are discussed below.

Fish Tissue Sampling Database

These risk and hazard estimates are based on 1998 to 2000 fish sampling data from the Housatonic River and it is assumed that the fish tissue concentrations will remain constant throughout the duration of the risk assessment. While these sampling data may be representative of current conditions in the river, concentrations may change over time, especially if individuals consume fish from the river for up to 90 years, as is possible in this MEE model. There are no site-specific trend data available to permit a quantification of PCB declines in fish tissue concentrations over time and therefore no means to model exposure levels that will occur in the future. In the event that declines in fish tissue PCB concentrations occur in the

future as potential upstream sources are removed, future exposures will be less than those that have been modeled in this MEE.

Fish Consumption Rates

The fish consumption rates used in the MEE are based on the Ebert et al. (1993) angler survey data for different waterbody types. To be consistent with the methodology used to collect data in that angler survey, river/stream consumption rates have been used for those reaches of the river that are limited in size and are commonly identified as portions of the river. Lake/pond consumption rates have been used to evaluate exposures to large, standing waterbodies that are commonly identified as lakes (i.e., Rising Pond and Lake Lillinonah/Lake Zoar).

Woods Pond presents a quandary because it is commonly called Woods Pond but is small and is also clearly understood among locals to be a portion of the Housatonic River. Thus, it could have been evaluated using either the river/stream consumption rate distribution or the lake/pond consumption rate distribution. Use of the lake/pond consumption rate distribution would have yielded somewhat different results because of the shape of that distribution is different from the shape of the river/stream distribution in that all the percentiles up to the 95th percentile are higher for the lake/pond distribution than the corresponding percentiles for the river/stream distribution but, above that level, the percentile values for the river/stream consumption rates are higher (e.g., the 97th percentile for rivers/streams is 117 g/day while the 95th percentile for lakes/ponds is 75 g/day) and the maximum is substantially lower (see Table 2). It is likely that the use of the lake/pond consumption rate distribution would have yielded somewhat higher central tendency risk estimates but lower upper bound risk estimates.

These consumption rate estimates are based on reported fish consumption behaviors by Maine anglers who fished multiple waterbodies of the types identified during the 1-year survey period. Thus, they likely overestimate consumption from single waterbodies or single reaches of a waterbody, as is being evaluated in the HHRA.

Finally, it should be noted that in the combined adult/child cancer risk analysis of the MEE, adult fish consumption rates have been used for all individuals regardless of their age. This is because the model, as currently designed, does not allow age-specific fish consumption rates to be selected and only permits one distribution of fish consumption rates to be used. This has likely substantially overestimated exposures for children between the ages of 1 and 10 years

because of the high consumption rates relative to the low body weights of these individuals. However, for the evaluation of cancer risk, which is averaged over a lifetime of exposure, it is unlikely to have made a substantial difference in the lifetime risk estimates. This issue did not affect the non-cancer calculations for adults and children as these analyses were run separately, each using the relevant consumption rate distribution.

Exposure Duration

This analysis uses residence time in Berkshire County as a basis for exposure duration. This assumption likely overestimates actual duration of exposure for most fish consumers because it is likely that there will be years when individuals live in Berkshire County but do not fish the Housatonic River. Some individuals may not begin to fish the Housatonic River when they commence their residence in Berkshire County but may begin to fish it at some later point. In addition, as individuals age they may stop fishing the Housatonic River, either because they stop fishing completely (due to illness or loss of interest) or because access to the Housatonic River is more difficult than access to other nearby waterbodies. Still other individuals, who are less avid anglers, may fish sporadically so that they fish the river during some years but not others. Finally, individuals who regularly participate in recreational fishing may fish a variety of local water bodies and thus do not necessarily fish the Housatonic River each year. In all cases, the assumption that residence time equates to time fishing the Housatonic River will tend to overestimate actual exposures that are likely to occur.

EPA's MEE used the results of the Massachusetts Department of Public Health (MADPH) survey to develop its exposure duration distribution. From that survey, EPA selected the information collected related to the number of years that individuals reported that they had eaten freshwater fish. Since the raw data for these distributions have not been provided, the only available information from the survey consists of summary statistics. However, a comparison of the summary statistics from the distribution used by EPA and that used by AMEC indicates that they are similar.

Comparison of Underlying Distribution for Exposure Duration

	MADPH Survey Data on Years Consuming Freshwater fish	AMEC Exposure Duration Distribution Based on Census Data
Minimum	1	1
Maximum	82	75
Mean	22.5	24.0
Median	20	20
95^{th} %ile	60	59

It should be noted that while the MADPH survey data had a maximum value of 82 years, EPA truncated that distribution to 70 years to be consistent with its definition of lifetime exposure. Thus, these two sources of information about potential exposure duration yielded similar results.

In addition, as discussed previously, Berkshire County census data have been used to estimate exposure duration for the Connecticut reaches of the river. While this may be appropriate for the more rural portions of northern Connecticut (near Reaches 11 and 12), it likely overestimates duration for Reaches 14 and 15, which are proximate to more urban areas and thus generally have higher mobility rates.

Toxicity Values

The point estimate toxicity values used in the MEE 1 analysis have substantial uncertainties associated with them. Because toxicity values based on human epidemiological evidence are not available for PCBs, the point estimate CSF and RfD values used in the MEE 1 model are based on the results of animal bioassay data and have been derived using a number of conservative assumptions and adjustment factors in an attempt to predict potential human response. Because of the conservatism introduced by the approaches used, it is likely that the actual toxicity of PCBs to humans has been overestimated. The MEE 2 analysis has attempted to characterize some of this uncertainty by substituting distributions of dose-response values for the point estimates used in MEE 1. There are still, however, uncertainties associated with these distributions because the underlying data are still based on the results of animal bioassays, and the weight of available human evidence concerning the toxicity of PCBs indicates that humans are not as sensitive as laboratory animals to the toxic effects of PCBs (see Attachments J and K

to this set of comments). Thus, the MEE 2 model also likely overestimates potential risks to humans.

Use of Subchronic Reference Dose for Young Children

As discussed above, this MEE analysis used EPA's subchronic RfD (or a distribution of subchronic RfDs) for children aged 1 to 6 years. Since the exposure durations for young children ranged from as little as one year to as many as six years, use of a subchronic RfD is consistent with EPA's (1989) definition for subchronic exposure (< 7 years). In the HHRA, however, EPA has applied a chronic RfD to these subchronic exposures as an added level of conservatism. Application of the chronic RfD (or a distribution of such RfDs) to the exposures calculated for children in this MEE would increase the respective HIs. For the MEE 1 analysis, for example, the HIs would increase by a factor of 2.5.

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Figure 1. Structure and Organization of the MEE Model for Housatonic River Anglers

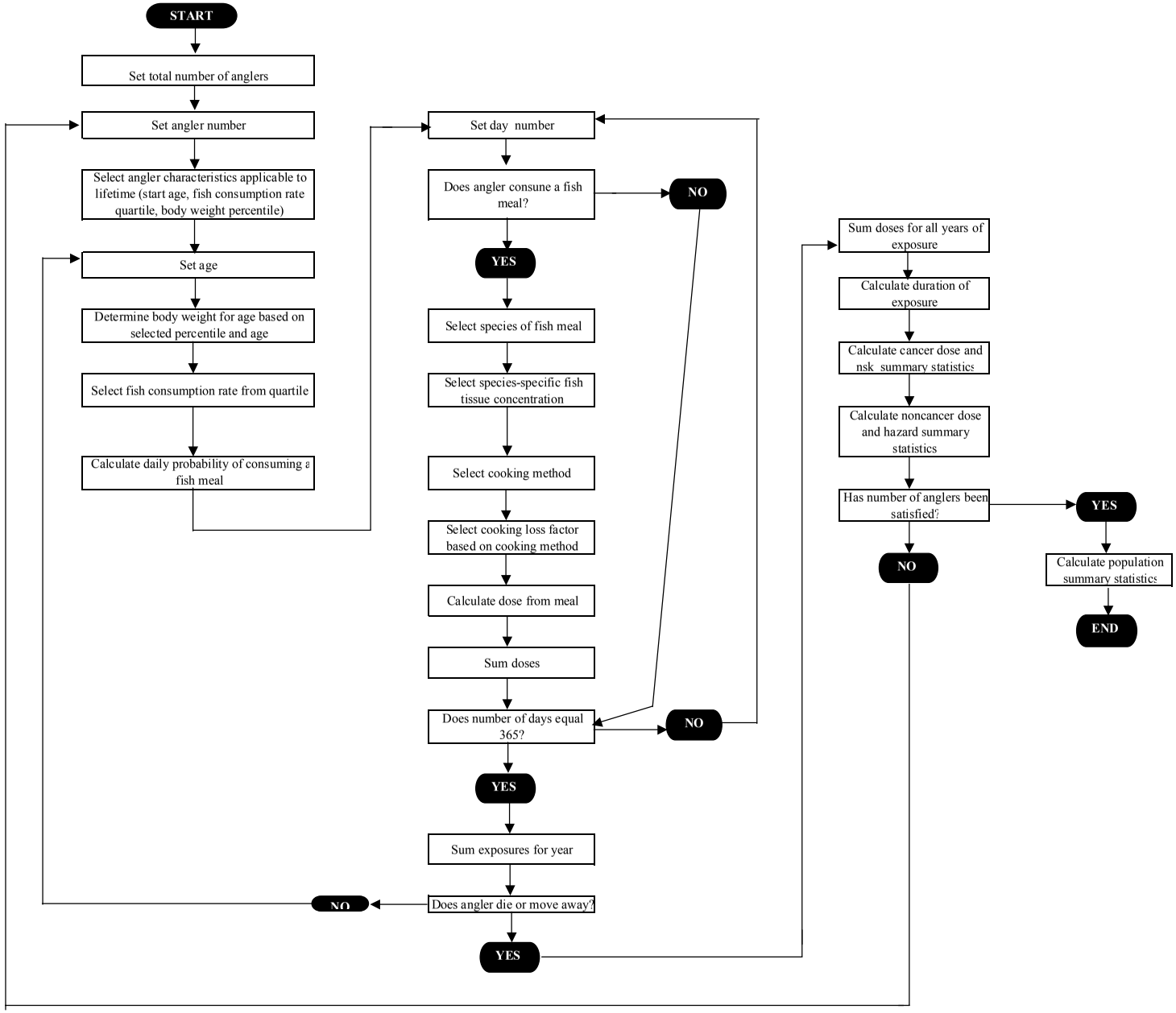


Table 1. Cumulative Start Age Distribution Based on Census Statistics for Each Age Group

Age (yrs)	Cumulative Start Age Probability Based on Population Size		Age (yrs)	Cumulative Start Age Probability Based on Population Size	
	Massachusetts[1]	Connecticut[2]		Massachusetts[1]	Connecticut[2]
1	0.01356	0.01756	46	0.63313	0.68429
2	0.02713	0.03511	47	0.64916	0.69959
3	0.04069	0.05267	48	0.66519	0.71489
4	0.05425	0.07022	49	0.68123	0.73019
5	0.06730	0.08539	50	0.69642	0.74406
6	0.08036	0.10056	51	0.71161	0.75793
7	0.09341	0.11573	52	0.72680	0.77180
8	0.10646	0.13089	53	0.74200	0.78567
9	0.11952	0.14606	54	0.75719	0.79954
10	0.13372	0.16083	55	0.76886	0.81034
11	0.14792	0.17560	56	0.78053	0.82113
12	0.16211	0.19037	57	0.79220	0.83193
13	0.17631	0.20514	58	0.80387	0.84273
14	0.19051	0.21990	59	0.81554	0.85352
15	0.20563	0.23268	60	0.82495	0.86157
16	0.22075	0.24546	61	0.83437	0.86962
17	0.23587	0.25824	62	0.84379	0.87767
18	0.25099	0.27101	63	0.85320	0.88572
19	0.26611	0.28379	64	0.86262	0.89377
20	0.27767	0.29473	65	0.87162	0.90096
21	0.28924	0.30566	66	0.88062	0.90814
22	0.30080	0.31660	67	0.88962	0.91533
23	0.31237	0.32753	68	0.89863	0.92252
24	0.32393	0.33847	69	0.90763	0.92970
25	0.33424	0.35073	70	0.91669	0.93666
26	0.34455	0.36299	71	0.92575	0.94362
27	0.35486	0.37525	72	0.93481	0.95058
28	0.36517	0.38751	73	0.94387	0.95754
29	0.37548	0.39977	74	0.95293	0.96450
30	0.38812	0.41516	75	0.96111	0.97069
31	0.40075	0.43055	76	0.96929	0.97689
32	0.41338	0.44594	77	0.97747	0.98308
33	0.42601	0.46133	78	0.98565	0.98928
34	0.43865	0.47672	79	0.99383	0.99547
35	0.45449	0.49448	80	1	1
36	0.47034	0.51224	81	1	1
37	0.48619	0.53000	82	1	1
38	0.50203	0.54776	83	1	1
39	0.51788	0.56552	84	1	1
40	0.53452	0.58315	85	1	1
41	0.55115	0.60078	86	1	1
42	0.56779	0.61842	87	1	1
43	0.58442	0.63605	88	1	1
44	0.60106	0.65369	89	1	1
45	0.61709	0.66899	90	1	1

1. Massachusetts data based on Berkshire County only.

2. Connecticut data based on Fairfield, Litchfield and New Haven Counties combined.

Table 2. Summary of Adult Fish Consumption Rates Used in the MEE Analysis

Percentile	Rivers and Streams Consumption Rates (g/day)	Lakes and Ponds Consumption Rates (g/day)
Number of data points	446	503
Minimum	0.023	0.014
5	0.13	0.2
10	0.18	0.3
15	0.25	0.39
20	0.32	0.50
25	0.41	0.68
30	0.54	0.89
35	0.64	1.0
40	0.73	1.3
45	0.87	1.5
50	1.0	1.7
55	1.3	2.2
60	1.5	2.5
65	1.7	2.8
70	2.1	3.3
75	2.6	4.1
80	3.6	5.5
85	4.4	6.8
90	6.1	9.7
95	12	16
Maximum	118	92

Table 3. Summary of Species-Specific Cooking Methods Probabilities Based on Responses to the Maine Angler Survey

	Fry	Bake	Broil/Grill	Poach/Boil/Soup	Raw
Bass	0.48	0.25	0.18	0.08	0.009
Bullhead	1	0	0	0	0
Perch	0.67	0.074	0.099	0.16	0
Sunfish	0.8	0	0.2	0	0
Trout	0.68	0.16	0.14	0.02	0.008

Table 4. Summary of Cooking Loss Factors for PCBs in Fish Tissues with Low Lipid Contents

Broil/Grill					
Author	**Cited in**	**Species**	**Prep. Method**	**Lipid Content**	**Percent Loss**
Daubenmire, 1996	HHRA Table 4-18	Lake trout	Skin-off fillet	Not listed	24
Daubenmire, 1996	HHRA Table 4-18	Lake trout	Skin-off fillet	Not listed	24
Daubenmire, 1996	HHRA Table 4-18	Lake trout	Skin-off fillet	Not listed	20
Daubenmire, 1996	HHRA Table 4-18	Lake trout	Skin-off fillet	Not listed	25
Zabik, 1996	HHRA Table 4-18	Lake trout	Skin-off fillet	Not listed	15
Zabik, 1996	HHRA Table 4-18	Lake trout	Skin-off fillet	Not listed	7
Zabik, 1996	HHRA Table 4-18	Lake trout	Skin-off fillet	Not listed	12
				Average Loss	**18%**
Bake					
Author	**Cited in**	**Species**	**Prep. Method**	**Lipid Content**	**Percent Loss**
Smith et al., 1973/Smith 1972	Scherer and Price, 1993	Chinook salmon	Skin-off fillet	2.70%	10
Skea et al., 1981	Scherer and Price, 1993	Smallmouth bass	Skin-on fillet	2.80%	16
				Average Loss	**13%**
Fried					
Author	**Cited in**	**Species**	**Prep. Method**	**Lipid Content**	**Percent Loss**
Daubenmire, 1996	HHRA Table 4-18	White bass	Skin-on fillet	Not listed	17
Daubenmire, 1996	HHRA Table 4-18	White bass	Skin-on fillet	Not listed	21
Skea et al., 1981	Scherer and Price, 1993	Smallmouth bass	Skin-on fillet	1.30%	74
Puffer and Gossett, 1983	Scherer and Price, 1993	White croaker	Skin-off fillet	1.20%	65
Puffer and Gossett, 1983	Scherer and Price, 1993	White croaker	Skin-off fillet	0.90%	28
Wang and Harrad, 2000	HHRA Table 4-18	Trout	Skin-off fillet	Not listed	26
Wang and Harrad, 2000	HHRA Table 4-18	Trout	Skin-on fillet	Not listed	25
				Average Loss	**37%**
Poach/Boil/Soup					
Author	**Cited in**	**Species**	**Prep. Method**	**Lipid Content**	**Percent Loss**
Daubenmire, 1996	HHRA Table 4-18	Lake trout	Skin-off fillet	Not listed	12
Daubenmire, 1996	HHRA Table 4-18	Lake trout	Skin-off fillet	Not listed	13
Zabik et al., 1996	HHRA Table 4-18	Lake trout	Skin-off fillet	Not listed	10
				Average Loss	**12%**

Table 5. Average Reduction of PCBs in fish by Various Cooking Methods

Cooking Method	Average Reduction on a PCB Mass Basis	Fraction of PCBs Remaining after Cooking
Raw	0	1.0
Broil/Grill	18 percent	0.82
Bake	13 percent	0.87
Fry	37 percent	0.63
Poach/Boil/Soup	12 percent	0.88

Table 6. Summary of Age-Specific Distribution of Female Body Weights[1]

Body Weights (kg)				Body Weights (kg)			
Body Weight Variability				Body Weight Variability			
Age	5th Percentile	50th Percentile	95th Percentile	Age	5th Percentile	50th Percentile	95th Percentile
1	8.8	10.7	13.4	46	48.5	65.5	96
2	10.8	12.7	15.9	47	48.5	65.5	96
3	11.7	14.7	18.4	48	48.5	65.5	96
4	13.7	16.7	21.1	49	48.5	65.5	96
5	15.3	19.0	26.6	50	48.5	65.5	96
6	17.0	21.3	29.6	51	48.5	65.5	96
7	19.2	23.8	34.0	52	48.5	65.5	96
8	21.4	27.5	36.5	53	48.5	65.5	96
9	22.9	29.7	48.4	54	48.5	65.5	96
10	25.7	34.5	49.6	55	48.6	65.2	95.1
11	29.8	40.3	60	56	48.6	65.2	95.1
12	32.3	45.4	60.5	57	48.6	65.2	95.1
13	35.4	49.0	76.3	58	48.6	65.2	95.1
14	40.3	53.1	75.2	59	48.6	65.2	95.1
15	44.0	53.3	76.6	60	48.6	65.2	95.1
16	44.1	55.6	76.8	61	48.6	65.2	95.1
17	44.5	58.4	81.8	62	48.6	65.2	95.1
18	46.6	58.0	82.9	63	48.6	65.2	95.1
19	46.6	58.0	82.9	64	48.6	65.2	95.1
20	46.6	58.0	82.9	65	47.1	64.8	91.3
21	46.6	58.0	82.9	66	47.1	64.8	91.3
22	46.6	58.0	82.9	67	47.1	64.8	91.3
23	46.6	58.0	82.9	68	47.1	64.8	91.3
24	46.6	58.0	82.9	69	47.1	64.8	91.3
25	47.4	60.9	93.5	70	47.1	64.8	91.3
26	47.4	60.9	93.5	71	47.1	64.8	91.3
27	47.4	60.9	93.5	72	47.1	64.8	91.3
28	47.4	60.9	93.5	73	47.1	64.8	91.3
29	47.4	60.9	93.5	74	47.1	64.8	91.3
30	47.4	60.9	93.5	75	47.1	64.8	91.3
31	47.4	60.9	93.5	76	47.1	64.8	91.3
32	47.4	60.9	93.5	77	47.1	64.8	91.3
33	47.4	60.9	93.5	78	47.1	64.8	91.3
34	47.4	60.9	93.5	79	47.1	64.8	91.3
35	49.2	63.4	98.9	80	47.1	64.8	91.3
36	49.2	63.4	98.9	81	47.1	64.8	91.3
37	49.2	63.4	98.9	82	47.1	64.8	91.3
38	49.2	63.4	98.9	83	47.1	64.8	91.3
39	49.2	63.4	98.9	84	47.1	64.8	91.3
40	49.2	63.4	98.9	85	47.1	64.8	91.3
41	49.2	63.4	98.9	86	47.1	64.8	91.3
42	49.2	63.4	98.9	87	47.1	64.8	91.3
43	49.2	63.4	98.9	88	47.1	64.8	91.3
44	49.2	63.4	98.9	89	47.1	64.8	91.3
45	48.5	65.5	96	90	47.1	64.8	91.3

1. Based on EPA (1997)

Table 7. Summary of Age-Specific Distribution of Male Body Weights[1]

Body Weights (kg)				Body Weights (kg)			
Body Weight Variability				Body Weight Variability			
Age	5th Percentile	50th Percentile	95th Percentile	Age	5th Percentile	50th Percentile	95th Percentile
1	9.6	11.7	14.4	46	50.8	79.0	105.3
2	11.1	13.5	16.5	47	50.8	79.0	105.3
3	12.9	15.4	19.1	48	50.8	79.0	105.3
4	14.1	17.6	22.2	49	50.8	79.0	105.3
5	16.0	19.4	25.4	50	50.8	79.0	105.3
6	18.6	22.0	30.1	51	50.8	79.0	105.3
7	19.7	24.8	33.9	52	50.8	79.0	105.3
8	20.4	27.5	39.1	53	50.8	79.0	105.3
9	24.0	30.2	43.1	54	50.8	79.0	105.3
10	27.2	34.8	53.4	55	59.9	77.7	102.3
11	26.8	37.3	61.0	56	59.9	77.7	102.3
12	30.7	42.5	67.5	57	59.9	77.7	102.3
13	35.4	48.4	69.9	58	59.9	77.7	102.3
14	41.0	56.4	77.0	59	59.9	77.7	102.3
15	46.2	60.1	81.3	60	59.9	77.7	102.3
16	51.4	64.4	91.2	61	59.9	77.7	102.3
17	50.7	65.8	88.9	62	59.9	77.7	102.3
18	56.8	72.0	99.5	63	59.9	77.7	102.3
19	56.8	72.0	99.5	64	59.9	77.7	102.3
20	56.8	72.0	99.5	65	54.4	74.2	96.6
21	56.8	72.0	99.5	66	54.4	74.2	96.6
22	56.8	72.0	99.5	67	54.4	74.2	96.6
23	56.8	72.0	99.5	68	54.4	74.2	96.6
24	56.8	72.0	99.5	69	54.4	74.2	96.6
25	59.5	77.5	102.7	70	54.4	74.2	96.6
26	59.5	77.5	102.7	71	54.4	74.2	96.6
27	59.5	77.5	102.7	72	54.4	74.2	96.6
28	59.5	77.5	102.7	73	54.4	74.2	96.6
29	59.5	77.5	102.7	74	54.4	74.2	96.6
30	59.5	77.5	102.7	75	54.4	74.2	96.6
31	59.5	77.5	102.7	76	54.4	74.2	96.6
32	59.5	77.5	102.7	77	54.4	74.2	96.6
33	59.5	77.5	102.7	78	54.4	74.2	96.6
34	59.5	77.5	102.7	79	54.4	74.2	96.6
35	59.7	79.9	104.3	80	54.4	74.2	96.6
36	59.7	79.9	104.3	81	54.4	74.2	96.6
37	59.7	79.9	104.3	82	54.4	74.2	96.6
38	59.7	79.9	104.3	83	54.4	74.2	96.6
39	59.7	79.9	104.3	84	54.4	74.2	96.6
40	59.7	79.9	104.3	85	54.4	74.2	96.6
41	59.7	79.9	104.3	86	54.4	74.2	96.6
42	59.7	79.9	104.3	87	54.4	74.2	96.6
43	59.7	79.9	104.3	88	54.4	74.2	96.6
44	59.7	79.9	104.3	89	54.4	74.2	96.6
45	50.8	79.0	105.3	90	54.4	74.2	96.6

1. Based on EPA (1997)

Table 8. Berkshire County Data for Mortality and Mobility (Out of County Moves)

	Mortality Probability		Probability of Moving	
Age (yrs)	Male	Female	Male	Female
1	0.001144	0.000577	0.030478	0.030478
2	0.001144	0.000577	0.030478	0.030478
3	0.001144	0.000577	0.030478	0.030478
4	0.001144	0.000577	0.030478	0.030478
5	0.000233	0	0.030478	0.030478
6	0.000233	0	0.030478	0.030478
7	0.000233	0	0.030478	0.030478
8	0.000233	0	0.030478	0.030478
9	0.000233	0	0.030478	0.030478
10	0.000212	0.000228	0.024014	0.024014
11	0.000212	0.000228	0.024014	0.024014
12	0.000212	0.000228	0.024014	0.024014
13	0.000212	0.000228	0.024014	0.024014
14	0.000212	0.000228	0.024014	0.024014
15	0.000803	0.000635	0.047191	0.047191
16	0.000803	0.000635	0.047191	0.047191
17	0.000803	0.000635	0.047191	0.047191
18	0.000803	0.000635	0.047191	0.047191
19	0.000803	0.000635	0.047191	0.047191
20	0.000793	0.000000	0.089978	0.089978
21	0.000793	0.000000	0.089978	0.089978
22	0.000793	0.000000	0.089978	0.089978
23	0.000793	0.000000	0.089978	0.089978
24	0.000793	0.000000	0.089978	0.089978
25	0.000922	0.000595	0.072214	0.072214
26	0.000922	0.000595	0.072214	0.072214
27	0.000922	0.000595	0.072214	0.072214
28	0.000922	0.000595	0.072214	0.072214
29	0.000922	0.000595	0.072214	0.072214
30	0.000762	0.000240	0.044017	0.044017
31	0.000762	0.000240	0.044017	0.044017
32	0.000762	0.000240	0.044017	0.044017
33	0.000762	0.000240	0.044017	0.044017
34	0.000762	0.000240	0.044017	0.044017
35	0.001606	0.000771	0.015802	0.015802
36	0.001606	0.000771	0.015802	0.015802
37	0.001606	0.000771	0.015802	0.015802
38	0.001606	0.000771	0.015802	0.015802
39	0.001606	0.000771	0.015802	0.015802
40	0.002325	0.001813	0.015802	0.015802
41	0.002325	0.001813	0.015802	0.015802
42	0.002325	0.001813	0.015802	0.015802
43	0.002325	0.001813	0.015802	0.015802
44	0.002325	0.001813	0.015802	0.015802
45	0.002580	0.003046	0.010472	0.010472
46	0.002580	0.003046	0.010472	0.010472
47	0.002580	0.003046	0.010472	0.010472
48	0.002580	0.003046	0.010472	0.010472
49	0.002580	0.003046	0.010472	0.010472
50	0.004370	0.002831	0.010472	0.010472
51	0.004370	0.002831	0.010472	0.010472
52	0.004370	0.002831	0.010472	0.010472
53	0.004370	0.002831	0.010472	0.010472
54	0.004370	0.002831	0.010472	0.010472
55	0.008688	0.006374	0.007763	0.007763
56	0.008688	0.006374	0.007763	0.007763
57	0.008688	0.006374	0.007763	0.007763
58	0.008688	0.006374	0.007763	0.007763
59	0.008688	0.006374	0.007763	0.007763
60	0.012719	0.007658	0.007763	0.007763
61	0.012719	0.007658	0.007763	0.007763
62	0.012719	0.007658	0.007763	0.007763
63	0.012719	0.007658	0.007763	0.007763
64	0.012719	0.007658	0.007763	0.007763
65	0.021607	0.012102	0.006429	0.006429
66	0.021607	0.012102	0.006429	0.006429
67	0.021607	0.012102	0.006429	0.006429
68	0.021607	0.012102	0.006429	0.006429
69	0.021607	0.012102	0.006429	0.006429
70	0.027273	0.017656	0.006429	0.006429
71	0.027273	0.017656	0.006429	0.006429
72	0.027273	0.017656	0.006429	0.006429
73	0.027273	0.017656	0.006429	0.006429
74	0.027273	0.017656	0.006429	0.006429
75	0.057971	0.032455	0.005800	0.005800
76	0.057971	0.032455	0.005800	0.005800
77	0.057971	0.032455	0.005800	0.005800
78	0.057971	0.032455	0.005800	0.005800
79	0.057971	0.032455	0.005800	0.005800
80	0.111276	0.062476	0.005800	0.005800
81	0.111276	0.062476	0.005800	0.005800
82	0.111276	0.062476	0.005800	0.005800
83	0.111276	0.062476	0.005800	0.005800
84	0.111276	0.062476	0.005800	0.005800
85	0.178683	0.159432	0.010167	0.010167
86	0.178683	0.159432	0.010167	0.010167
87	0.178683	0.159432	0.010167	0.010167
88	0.178683	0.159432	0.010167	0.010167
89	0.178683	0.159432	0.010167	0.010167
90	0.178683	0.159432	0.010167	0.010167

Table 9. Connecticut Data for Mortality in Litchfield, New Haven and Fairfield Counties Combined

	Mortality Probability			Mortality Probability	
Age (yrs)	Male	Female	Age (yrs)	Male	Female
1	0.001622	0.000913	46	0.003074	0.001839
2	0.001622	0.000913	47	0.003074	0.001839
3	0.001622	0.000913	48	0.003074	0.001839
4	0.001622	0.000913	49	0.003074	0.001839
5	0.000085	0.000104	50	0.004469	0.002929
6	0.000085	0.000104	51	0.004469	0.002929
7	0.000085	0.000104	52	0.004469	0.002929
8	0.000085	0.000104	53	0.004469	0.002929
9	0.000085	0.000104	54	0.004469	0.002929
10	0.000189	0.000107	55	0.007147	0.004463
11	0.000189	0.000107	56	0.007147	0.004463
12	0.000189	0.000107	57	0.007147	0.004463
13	0.000189	0.000107	58	0.007147	0.004463
14	0.000189	0.000107	59	0.007147	0.004463
15	0.000600	0.000338	60	0.014097	0.008063
16	0.000600	0.000338	61	0.014097	0.008063
17	0.000600	0.000338	62	0.014097	0.008063
18	0.000600	0.000338	63	0.014097	0.008063
19	0.000600	0.000338	64	0.014097	0.008063
20	0.001442	0.000444	65	0.022936	0.014647
21	0.001442	0.000444	66	0.022936	0.014647
22	0.001442	0.000444	67	0.022936	0.014647
23	0.001442	0.000444	68	0.022936	0.014647
24	0.001442	0.000444	69	0.022936	0.014647
25	0.001223	0.000458	70	0.033256	0.022166
26	0.001223	0.000458	71	0.033256	0.022166
27	0.001223	0.000458	72	0.033256	0.022166
28	0.001223	0.000458	73	0.033256	0.022166
29	0.001223	0.000458	74	0.033256	0.022166
30	0.001377	0.000613	75	0.053933	0.034569
31	0.001377	0.000613	76	0.053933	0.034569
32	0.001377	0.000613	77	0.053933	0.034569
33	0.001377	0.000613	78	0.053933	0.034569
34	0.001377	0.000613	79	0.053933	0.034569
35	0.001934	0.000904	80	0.085831	0.057148
36	0.001934	0.000904	81	0.085831	0.057148
37	0.001934	0.000904	82	0.085831	0.057148
38	0.001934	0.000904	83	0.085831	0.057148
39	0.001934	0.000904	84	0.085831	0.057148
40	0.002446	0.001245	85	0.155260	0.128651
41	0.002446	0.001245	86	0.155260	0.128651
42	0.002446	0.001245	87	0.155260	0.128651
43	0.002446	0.001245	88	0.155260	0.128651
44	0.002446	0.001245	89	0.155260	0.128651
45	0.003074	0.001839	90	0.155260	0.128651

Table 10. Distribution of Toxicity Values Used in the MEE 2 Analysis

Percentile	Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Chronic Reference Dose (ng/kg-day)	Subchronic Reference Dose (ng/kg-day)
0.1	0.40	18	38
1	0.40	31	66
5	0.41	59	119
10	0.42	84	164
15	0.43	103	201
20	0.44	123	239
25	0.45	142	276
30	0.46	163	313
35	0.47	184	349
40	0.48	205	386
45	0.49	225	422
50	0.50	246	459
55	0.64	277	511
60	0.78	307	563
65	0.92	338	615
70	1.06	369	667
75	1.20	400	718
80	1.27	465	820
85	1.35	530	922
90	1.42	595	1,023
95	1.50	734	1,228
99	2.06	1,086	1,717
99.9	2.19	1,716	2,500
100	2.20	1,786	2,587

EXHIBIT H.2

DISTRIBUTIONS OF TOXICITY DOSE-RESPONSE VALUES FOR PCBS

EXHIBIT H.2

Distributions of Toxicity Dose-Response Values for PCBs

This Exhibit describes the basis and methodology for the development of the distributions of the PCB toxicity values – i.e., Cancer Slope Factors (CSFs) and non-cancer Reference Doses (RfDs) – used by AMEC in its second alternative Microexposure Event (MEE 2) model of fish consumption risks, which is discussed in detail in Exhibit H.1.

Distribution of Cancer Slope Factors

EPA traditionally evaluates carcinogens by first assigning a weight-of-evidence classification, and then calculating a CSF that quantitatively defines the relationship between dose and response (EPA, 1989). The weight-of-evidence classification is typically based on the amount and quality of evidence that a compound is carcinogenic in humans and/or in experimental animals. The weight-of-evidence process requires an evaluation of available studies and their relative merits, while the development of a CSF requires the utilization of the data from those studies to develop an estimate of the probability of a carcinogenic response per unit intake of a chemical over a lifetime.

Past EPA risk assessments have been based on point estimates. In 2001, EPA issued a guidance document recognizing that probabilistic analysis tools are acceptable provided that risk assessors provide adequate supporting data and use credible assumptions in their work (EPA, 2001). That guidance provides the groundwork for employing these advanced techniques in exposure assessment. Similar techniques can be applied to toxicity assessment, resulting in more realistic risk estimates. In the case of PCBs, it is possible to move from the use of a point-estimate value that represents the cancer potency of PCBs to the use of a range of plausible CSFs for PCBs.

In 1996, EPA reevaluated the cancer potency and CSF estimates for PCBs, based on a number of studies involving both male and female rats (EPA, 1996).[1] Table 1 reports the central and upper-bound slope estimates derived in each of these studies, as reported by EPA (1996). In

[1] This reevaluation included the following studies: Brunner et al. (1996) (Aroclors 1016, 1242, 1254, 1260 for both male and female Sprague-Dawley rats), Kimbrough et al. (1975) (Aroclor 1260 for female Sherman rats), NCI (1978) (Aroclor 1254 for both male and female Fischer rats), Schaeffer et al. (1984) (A30 and A60 for male Wistar rats), and Norback and Weltman (1985) (Aroclor 1260 for both male and female Sprague-Dawley rats).

its reassessment, EPA (1996) recognized that the chronic rat feeding study of four PCB mixtures (Aroclors 1016, 1242, 1254, and 1260) performed by Brunner et al. (1996) and later published by Mayes et al. (1998) "provides the most comprehensive information for empirical modeling" (EPA, 1996, p. 32), and thus it used that study as the primary basis for developing its recommended range of CSFs.

Table. 1 Human Potency and Slope Estimates Derived from Rat Liver Tumors

Study, sex and strain, mixture	Central Slope $(mg/kg\text{-}d)^{-1}$	Upper-bound Slope $(mg/kg\text{-}d)^{-1}$
Brunner, F Sprague-Dawley, 1260	0.4	0.5
Brunner, F Sprague-Dawley, 1254	1.2	1.5
Brunner, F Sprague-Dawley, 1242	0.3	0.4
Brunner, F Sprague-Dawley, 1016	0.04	0.07
Brunner, M Sprague-Dawley, 1260	0.1	0.2
Brunner, M Sprague-Dawley, 1254	0.06	0.1
Brunner, M Sprague-Dawley, 1242	0.03	0.08
Brunner, M Sprague-Dawley, 1016	0.02	0.04
Kimbrough, F Sherman, 1260	1.0	1.1
NCI, M Fischer, 1254	0.1	0.2
NCI, F Fischer, 1254	0.08	0.2
Schaeffer, M Wistar, A 30	0.05	0.1
Schaeffer, M Wistar, A 60	1.7	2.1
Norback, M Sprague-Dawley, 1260	0.1	0.2
Norback, F Sprague-Dawley, 1260	1.6	2.2

Because Aroclors 1254 and 1260 most closely resemble the PCB mixtures present at the Housatonic River site, and because they were shown to be the most potent, the CSF distribution for this analysis was developed using the data for these two Aroclors. In addition, because female rats appear to be more sensitive than male rats, only female rat data were considered in order to provide an additional layer of conservatism to the approach. Further, consistent with EPA's (1996) selection of CSF ranges, the CSF distribution was based primarily on the potencies observed for these Aroclors in female rats in the Brunner et al. (1996) study. The highest upper-bound CSF from this study was 1.5 per mg/kg-day for Aroclor 1254, while Aroclor 1260 had an upper-bound CSF of 0.5 per mg/kg-day. Similar to EPA's (1996) ranges, AMEC bounded the upper end of the distribution by the highest observed upper-bound slope – 2.2 per mg/kg-day, derived from female rats in the Norback and Weltman (1985) study. As a conservative approach, the lower end of the distribution was bounded by the central estimate slope for Aroclor 1260 of 0.4 per mg/kg-day (Brunner et al., 1996). The CSFs that were

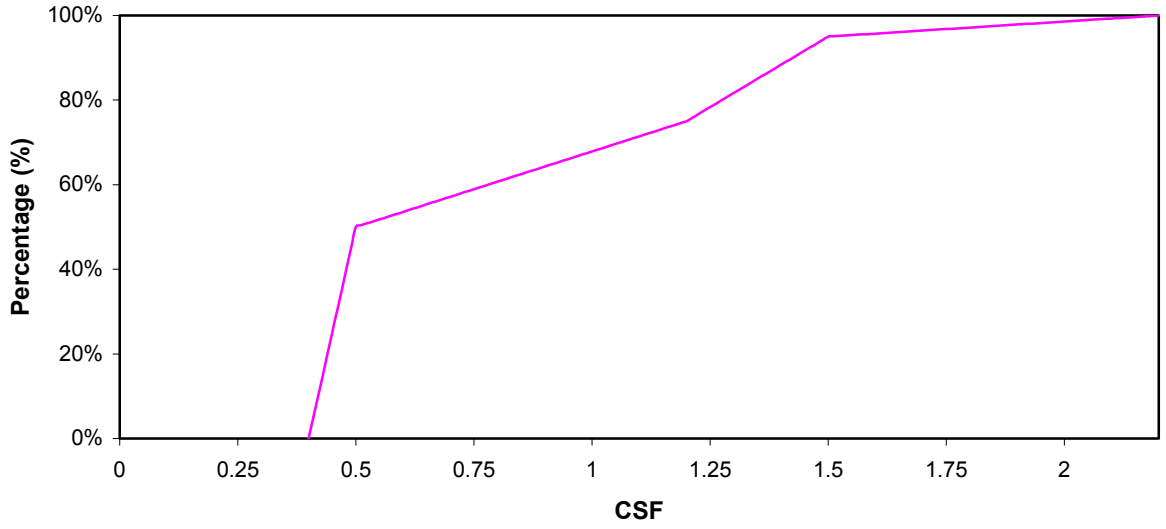
observed in other studies of Aroclors 1254 or 1260 fall within the range of this distribution, with the exception of the those studies that were discounted and given little weight by EPA (1996, p. 34).

Due to the reported Aroclor mixtures at the site, the upper-bound slope estimate of 0.5 for Aroclor 1260 was selected as the median value for the distribution. The central slope estimate of 1.2 for Aroclor 1254 was positioned at the 75th percentile while the upper-bound slope estimate of 1.5 for Aroclor 1254 was placed at the 95th percentile. The upper-bound slope estimate of 2.2 from the Norback and Weltman (1985) study became the maximum value in the distribution. Once these values were assigned their associated percentiles, a cumulative distribution was developed using the Palisades Corporation software @Risk version 4.5. The resulting distribution from 10,000 iterations is shown in Table 2 and graphically in Figure 1.

Table 2. Distribution of Cancer Slope Factors for PCBs

Percentile	CSF (mg/kg-d)$^{-1}$	Percentile	CSF (mg/kg-d)$^{-1}$
0	0.40	70	1.06
0.1	0.40	75	1.20
1	0.40	80	1.27
2	0.40	85	1.35
5	0.41	90	1.42
10	0.42	91	1.44
15	0.43	92	1.45
20	0.44	93	1.47
25	0.45	94	1.48
30	0.46	95	1.50
35	0.47	96	1.64
40	0.48	97	1.78
45	0.49	98	1.92
50	0.50	99	2.06
55	0.64	99.9	2.19
60	0.78	100	2.20
65	0.92		

Figure 1. CSF Distribution



This CSF distribution has been used in AMEC's alternative MEE 2 analysis described in Exhibit H.1.

Distribution of Reference Doses

EPA traditionally evaluates non-carcinogenic risks by comparing estimated dose rates with a point estimate RfD. The measure of risk is the ratio of the predicted dose to the RfD. If the ratio (called the hazard index) is less than one, then the dose is less than the RfD and no risk is predicted.

Generally, an RfD is based on the "no observed adverse effect level" (NOAEL) in the population of test animals studied and further lowered by a combination of uncertainty factors (UF) and modifying factors (MF), using the following equation:

$$\frac{NOAEL}{UF \times MF}$$

The UF is a composite uncertainty factor representing multiple uncertainty factors, and a MF is a situation-specific modifying factor. The uncertainty factors in the RfD include factors to express inter-species variation (UF_A), inter-individual variation (UF_H), LOAEL to NOAEL extrapolation (UF_L), subchronic to chronic extrapolation (UF_S), and database uncertainty (UF_D). Each uncertainty factor can be defined as a loose approximation of the upper bound of the

distribution of dose ratios associated with different toxicological endpoints and chemicals (Dourson, 1994; Swartout et al., 1998). Uncertainty factors have historically been assigned values of either 10 or 3 by EPA.

The establishment of an RfD requires the consideration of both variability and uncertainty in the estimate of human toxicological responses to chemicals. Variability in human response must be addressed in order to derive an estimate of a dose that is sufficiently low so as to be protective of individuals who are particularly sensitive to a compound. Uncertainty must be addressed because estimates of threshold in humans, which are based on NOAELs in test-animal studies, require a number of imprecise and uncertain extrapolations. Thus, the RfD must be set low enough such that there is little chance that it will be above the true threshold. As a result, the RfD can be thought of as the "lower confidence limit of a NOAEL in sensitive humans" (Swartout et al., 1998). This definition implies that the RfD is close to the lower-bound value of a range of doses that could be protective and that the actual level that is protective is likely to be higher than the RfD.

Recently, a number of authors have investigated how to characterize the uncertainty in the population threshold using the framework for setting RfDs (Baird et al., 1996; Slob and Pieters, 1997; Swartout et al., 1998). There is agreement that if the existing values of uncertainty factors are replaced with distributions that reflect inter-chemical variation in the appropriate ratios, the result will be an uncertainty distribution for the threshold of a compound (Swartout et al., 1998). This distribution should not be viewed as a representation of the uncertainty in the RfD, but rather as an estimate of the true but unknown threshold where the RfD is some point on the lower end of the distribution. In 1999, EPA's Science Advisory Panel under FIFRA recommended the use of such techniques for the evaluation of uncertainty in risk assessments, noting that "[a] distributional approach to non-cancer risk analysis would resolve the dilemma [that point-estimate uncertainty factors are hard to interpret] by specifying the whole distribution of the factors in question" (EPA, 1999, p. 45).

Swartout et al. (1998) described a probabilistic approach to quantifying uncertainty factors based on the definition and use of RfDs by the EPA. The approach does not attempt to distinguish one uncertainty factor from another based on empirical data or biological mechanisms, but rather uses a simple displaced lognormal distribution as a generic representation of all uncertainty factors. This reference distribution (U_R) is based on the existing concepts underlying EPA's current system of uncertainty factors (i.e., that the uncertainty value

of 10 is a loose upper-bound estimate of uncertainty and that no uncertainty factor is less than 1). Using these concepts and the tenet that toxicological data are generally lognormally distributed, Swartout et al. (1998) proposed a three-parameter lognormal distribution. Such a distribution is a standard two-parameter lognormal distribution that is shifted on the x-axis, starting at a value other than zero (i.e., offset). The three parameters are the mean (μ), the standard deviation (δ), and the offset (τ) and are set such that the median (50^{th} percentile) is $10^{0.5}$ and the 95^{th} percentile is 10 (Swartout et al., 1998). As stated previously, the 10 represents the high-end estimate of uncertainty and the choice of $10^{0.5}$ for the median is based on the common use of the value of 3 as an alternate uncertainty factor. The mean is equal to the logarithm of the offset-adjusted median of U_R [$\mu = \log_{10}(\text{median}(U_R) - \tau)$]. The parameter values satisfying these assumptions are $\mu = 0.335$, $\delta = 0.3765$ and $\tau = 1$. In cases where an uncertainty factor of 3 represents the loose upper-bound estimate of uncertainty, a simple approximation of the distribution is the square root of U_R (Swartout et al., 1998).

The methodology of Swartout et al. (1998) has been applied to EPA's selection of uncertainty factors for the Agency's chronic PCB RfD (used in the HHRA) to develop an uncertainty distribution of the population threshold for PCBs. The current chronic RfD for PCB Aroclor 1254 cited in EPA's IRIS database (EPA, 2003) and used in the HHRA is 20 ng/kg-day. The point-estimate uncertainty factors applied by EPA in deriving that RfD are described in Attachment N to this set of comments. To develop a distribution of RfDs, the equations for setting an RfD were used but the point-estimate uncertainty factors were replaced with distributions. A probabilistic technique (Monte Carlo Analysis with Latin Hypercube) was then used to determine the uncertainty in the estimate of the population threshold. The results are given in Table 3 and shown graphically in Figure 2.

As demonstrated by Table 3, the most likely estimate of the population threshold (50^{th} percentile of 246 ng/kg-day) is 12 times higher than EPA's current RfD (20 ng/kg-day), which falls at the lowest end of the distribution.

A similar distribution of RfDs was developed for subchronic exposures. The methodology was identical to that used to develop the distribution of chronic RfDs for PCBs, except that no uncertainty factor was necessary to extrapolate from subchronic to chronic exposures. The resulting distribution of subchronic RfDs is shown in Table 4.

Table 3. Distribution of Chronic Reference Doses	
Percentiles	**RfD (ng/kg-day)**
0.1	18
1	31
5	59
10	84
15	103
20	123
25	142
30	163
35	184
40	205
45	225
50	246
55	277
60	307
65	338
70	369
75	400
80	465
85	530
90	595
95	734
99	1086
100	1786

Figure 2. Chronic RfD Distribution

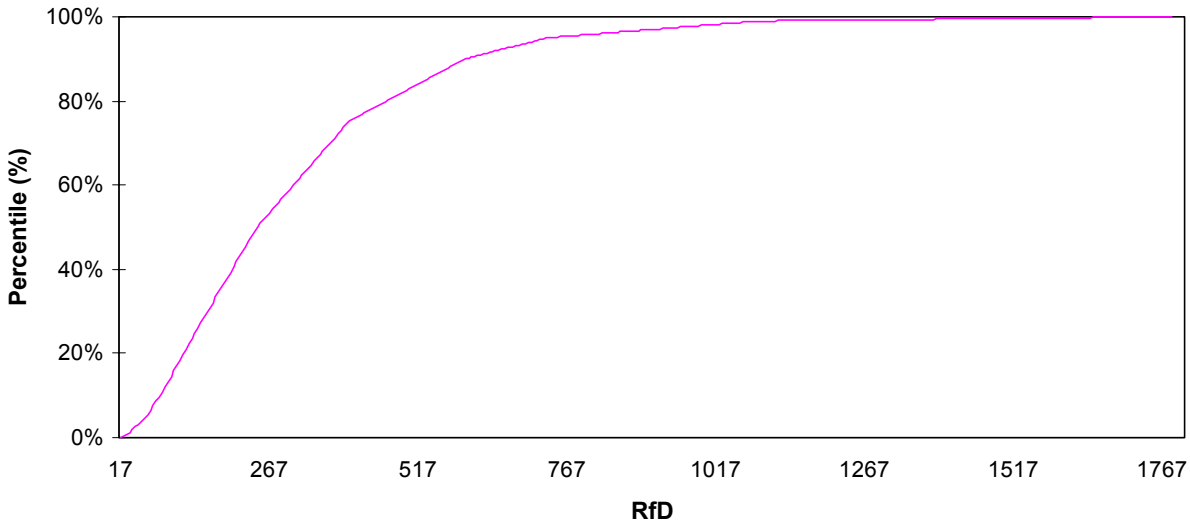


Table 4. Distribution of Subchronic Reference Doses	
Percentiles	**RfD (ng/kg-day)**
0.1	38
1	66
5	119
10	164
15	201
20	239
25	276
30	313
35	349
40	386
45	422
50	459
55	511
60	563
65	615
70	667
75	718
80	820
85	922
90	1,023
95	1,228
99	1,717
100	2,587

The above findings demonstrate that the use of the current RfD likely overestimates the potential for non-carcinogenic risks. The use of distributions of RfDs, as derived above, can account for the uncertainty associated with the RfD. These distributions have been used for characterizing non-cancer risks in AMEC's alternative MEE 2 model, in which variabilities of both exposure parameters and toxicological factors are considered (see Exhibit H.1).

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